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NERVOUS AND MENTAL DISEASES

BY

ARCHIBALD CHURCH, M.D.
Professor of Nervous and Mental Diseases and Medical Jurisprudence in the Northwestern University
Medical School (the Chicago Medical College), Chicago; Late Professor of Neurology
in the Chicago Policlinic; Neurologist to St. Luke's, Wesley, and
Mercy Hospitals; Consulting Neurologist to the Home
for Destitute Crippled Children, Chicago, etc.

AND

FREDERICK PETERSON, M.D.
President of the State Commission in Lunacy, New York; Clinical Professor of Neurology and Psychiatry,
Columbia University; Consulting Alienist, Bellevue Hospital; Ex-President of the
New York Neurological Society; General Consultant to the
Craig Colony for Epileptics.

WITH 341 ILLUSTRATIONS

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PREFACE TO THE FIFTH EDITION.

In the preparation of this edition the section on Nervous Diseases has been carefully reviewed in order to place it abreast of the current literature on the subject, rather than for the purpose of making any material changes in the arrangement or descriptive text. This has necessitated a very large number of brief interpolations, which without materially enlarging the section, it is thought decidedly improves its subject matter. Several old cuts have been withdrawn and new ones inserted.

In the section on Insanity, the classification of Kraepelin has been added to the chapter on classifications for purposes of reference, and two new chapters have been written for the section upon Manic-Depressive Insanity and on Dementia Præcox, respectively. These additions with some minor changes here and there serve to place this section in line with recent progress in psychiatry.

The continued favor with which the work is received, as attested by this early opportunity for a fifth edition, is a source of great satisfaction to the authors.

August, 1905.

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PREFACE.

THIS book has been written for medical students and general practitioners. It makes no claim to be other than a carefully prepared text-book. The literature of neurology and psychiatry has been sifted by the authors, and such digest revised in the light of their own experience in practice and in teaching. They have attempted to present their facts clearly, directly, and with brevity, despite the difficulty of condensing two great subjects within the limits of a single volume.

This is not the joint work of two writers, but each author—Dr. Church in Neurology, and Dr. Peterson in Psychiatry—has contributed to the making of a single volume what might have made a separate monograph; each is, therefore, solely responsible for the work in his own department. In placing the correlated sciences neurology and psychiatry under the same cover, the reader's convenience was considered.

An unusual number of illustrations for each department (from the authors' own material, except when otherwise indicated) has been allowed by a generous publisher.

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NERVOUS DISEASES.

BY

ARCHIBALD CHURCH, M. D.

NERVOUS DISEASES.

PART I.

EXAMINATION OF PATIENTS.

CHAPTER I.

THE ANAMNESIS.

Diagnostic investigation in neurological work is a fine art. A correct opinion depends upon it. It is the first step toward treatment, the legitimate end of medicine. The physician is dealing with morbid conditions, revealing themselves for the most part by functional errors. The large subjective side of the problem, with the unreliability of the patient's responses, adds to the difficulty and calls for keenness of judgment at every step. The use of several tests for determining a given state is of much importance, especially in conditions that are not objectively evident. By comparison of the results thus obtained, and sometimes by striking an average of such results, exactness may be approximately reached, while dependence upon any one of them might be misleading.

On the other hand, by the use of instruments of precision, controlled by anatomical and physiological rules, a definiteness can often be reached in nervous diseases not elsewhere possible. Localization in brain and cord lesions is sometimes exact to a degree, and prognosis is often absolutely clear. But in many instances of the so-called functional diseases, careful study, the shrewdest judgment, and a wide experience enable one only approximately to appreciate the situation. The entire data can be secured only by a systematic and frequently prolonged or often-repeated examination, and it is of the first importance that the medical man should maintain an entirely judicial and non-committal mental attitude toward his patient and the diagnosis until he has every available fact at his disposal. Snap-shot diagnoses may be gratifying to all concerned, if correct, but they are very likely to be wrong and prejudicial to a proper subsequent estimation of the case and are never necessary.

A systematic examination can not be made without a prearranged formula. The nearer this corresponds to the development of the case, the less likely are important matters to be overlooked. It therefore should be chronological. Most patients insist on telling their own stories in their own way. It is sometimes well, especially in private practice, to allow them to do so, and when they finish, to begin properly. In nervous diseases the family history is often of paramount importance. Taken with the appearance of the patient, it gives valuable indications as to the constitutional make-up of the individual. The family history, then, is to be investigated first, after noting the *name*, *age*, *sex*, *nationality*, *occupation*, and *social state* of the patient.

Neurotic Heredity.—In seeking information regarding the antecedents of a patient, much tact must sometimes be employed. Patients are loath to detail matters of this character, not always from a wish to conceal them, but from disinclination to admit even to themselves any serious shortcoming or morbidity. To the half that is directly learned an equal amount may sometimes be reasonably added. Much can be learned by interrogating other members of the family, especially if related by marriage, the family physician, and old acquaintances, but the confidence and rights of the patient must not be forgotten. Nor is it sufficient to seek for instances of the identical disease in the family history of the case. The significant factor is a neuropathic liability, and this is indicated with more or less force by the appearance of nervous and mental diseases of any sort, of slight or serious degree, in the ascendants. For instance, a hysterical mother has an epileptic child and an idiotic grandchild; or highly wrought nervous organizations in mother and father eventuate in neurasthenic and unstable children. *Atavism* in mental and nervous diseases is quite common. *Insanity* in the same or neighboring generations may alternate with neuroses or mild psychoses, and any combination may exist. *Consanguinity* on the part of parents, in addition to furnishing a tendency to infecundity, is likely to bring together the subjects of similar neurotic taints, which are thereby reinforced in the offspring. In itself, however, consanguinity has probably been overestimated as a causal factor in nervous and mental diseases. The association of crime, precocity, genius, neuroses, and psychoses in related individuals may be encountered. Certain nervous diseases are of direct hereditary character, being transmitted from generation to generation or appearing in a group of cases in a given generation. It is only needful to mention Friedreich's ataxia and Huntingdon's chorea, but tabes dorsalis, paralysis agitans, multiple sclerosis, muscular atrophies, and practically every organic and functional nervous disease occasionally presents family groups of this nature.[1] Many of the familial diseases which have been described as essentially distinct are found to insensibly merge through the observation of intermediate cases; and Baumlin[2] contends that all typical diseases of a familial character drift into each other through such connecting links. A family constitutional defect is the only essential feature.

Debilitating diseases, like tuberculosis, rheumatism, and gout, are significant. Indeed, Charcot was inclined to consider arthritism as a congener of nervous diseases. *Inherited syphilis* is on an entirely different footing. Not only is it capable of producing embryological defects, but its blight may make itself evident on the part of the nervous apparatus during the period of growth or in adult life, modifying cerebral or spinal functions and at times leading to histological changes in the central and peripheral parts, which may vary in degree up to destructive lesions. *Diabetes* and *Bright's disease* are very common in neurotic families.

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[1] Ch. Féré, "La Famille Nevropathique," Paris, 1894.

[2] "Deut. Zeit. f. Nervenheilk.," Oct., 1901.

Personal History.—The investigator should go most carefully into the medical life-history of his patient. While doing so, indeed, whenever opportunity offers, the *conduct*, *attitude*, *manner*, *gait*, *posture*, *complexion*, *expression*, *gestures*, and *individuality* of the person should be keenly watched. This observation becomes in time a trained, almost automatic, faculty, so that minute details subconsciously apprehended at the time can be readily recalled.

In infancy, was there any birth difficulty, possible brain or spinal injury from protracted labor, precipitate labor, or instrumentation; lack of vigor, suspicion of syphilis, or convulsions? During *childhood*, did the patient present any nervous phenomena, such as marked delirium or spasms under febrile conditions or from irritation of the gums and intestinal tract? Was there enuresis, chorea, somnambulism, or night-terrors? Was he precocious or otherwise, docile or obstinate, cheerful or morose, forward or retiring? At *pubescence*, were there mental changes of unusual character, moodiness, expansiveness, cruelty? Was the establishment of menstruation attended by pain or hysterical manifestations? Was masturbation indulged in or suspected? During *adolescence*, what was the career, relation to the opposite sex, success in school and business, and what has been the course of events through *adult life?*

The past illnesses of the patient should then receive attention in the same systematic manner. The fevers and febricula of childhood, the exanthemata and infections. The diseases of the nasopharynx, stomach, intestines, and rectum, of the lungs and heart, of the skin, the special disorders of chest, abdomen, pelvis, and genito-urinary apparatus must not escape attention. Especial inquiry should be made for rheumatism, gout, grip, and malaria. *The venereal history* of the patient and the possibility of specific infection should in every case be carefully inquired into. This is a rule that has no exceptions. The investigation must be modified and guarded according to individual circumstances, but nothing should deter the physician from making sure that a factor of this sort is not overlooked. If *injuries* have been received, what were their character and consequences? Regarding *habits*, it is to be borne in mind that neurotic people are especially liable to carry everything to extremes and are prone to become the abject subjects of some perverted practice or stimulant addiction. Masturbation and venery take firm hold on them. Tobacco, alcohol, morphin, cocain, chloral, and even tea and coffee master them completely. These in turn often break down the moral status of the individual and make him unreliable and untruthful. Only inadvertently or at second hand does the physician sometimes gain the required information, but justifiable suspicion once aroused, he can usually go to the root of the matter.

In neurasthenic, hypochondriac, and hysterical cases frequently the patient has most seriously overestimated some such habit, is morbidly apprehensive as to its results, exaggerates its every relation, and the entire life seems to revolve around this central erroneous idea. One must avoid being led by such unfortunates into adopting their point of view. The details, duration, and probable effect of the habit being clearly understood, its morbific influence can be properly estimated. Let it never be forgotten that many of these habits are symptoms, not causes, of nervous disturbance, and are the result of an underlying predisposition upon which they are grafted. There they take on a morbid development and, in turn, no doubt, add to the unbalance of the individual.

The residence or habitat of the patient is an important consideration. It calls attention to the climatic and local conditions favoring health or disease. Unhealthful surroundings are of immediate concern, and throw a strong light upon the causation of many nervous disorders.

Occupation.—Many occupations predispose to nervous maladies and sometimes furnish the cause. Indeed, a group of diseases is known as occupation neuroses, of which writers' cramp is a type. Workers in metals, particularly those handling lead, phosphorus, mercury, and arsenic; people subjected to illuminating gases or bisulphid of carbon, and those who deal in alcoholics, and who are thereby likely to over-indulge, are subject to neuritides and associated mental disturbances. Work requiring exposure to cold and conditions favoring rheumatic processes entail a tendency to cerebral arterial mischief and peripheral palsies. Divers and those working in caissons, or elsewhere, under increased atmospheric pressure frequently present spinal lesions with paraplegic symptoms. Occupations which demand constant mental strain and sedentary habits, as in speculative mercantile life, teaching, and some of the professions, furnish large numbers of neurasthenics. On the other hand, the unoccupied are likely to become selfish, introspective, hysterical, and hypochondriac.

The Illness.—From the patient's statement, his personal history, and the physician's observation, the noting of the details of the illness under consideration is often a simple matter. The medical man from his special knowledge must supplement the impressions of the layman. In the "rheumatism" preceding ataxia he discerns initial features of tabes, and in some long-antecedent moral shock he recognizes the origin of the fixed hysterical idea that may have eventuated in a contracture. For each major group of nervous maladies, psychic, cerebral, spinal, and peripheral, he must follow out the clues his training recognizes or his studies and experience suggest.

Beginning with initial symptoms and alleged, suspected, or positively known causal conditions, the manifestations of the disease are to be systematically, briefly, and clearly developed and noted, with full attention to remissions, intermissions, or relapses. In convulsive disorders a full clinical investigation of the attack is of prime importance. It should embrace its exciting cause, onset, features of consciousness, motor signs and phenomena, attitudes, duration, termination, and sequelæ. In sensory disturbances investigate the particular dysesthesia or pain, its onset, exact location, intensity, duration, and associated conditions. In paralytic maladies determine the mode of onset, exact distribution, and the character and degree of motor failure. The mental symptoms are to be noted with equal care, attention being directed to their fixity, emotional character, and reasonableness or complete opposition to facts within the patient's range of knowledge. An important question is whether the patient can correct his morbid ideas by mental effort or is entirely subjugated by his delusional states. Finally, the tendency to improvement or the reverse should be indicated.

Physiognomy of the Patient.—Formerly it was considered sufficient to describe a patient as of the nervous, lymphatic, or sanguine temperament, and this has a certain value, but a better conception of the physical status is to be had from a study of the physiognomy of the individual. Based upon embryological conditions and formulated with some precision by Lombroso, Dejerine, Weismann, Fèrè, and others, we can recognize a type of degenerate or, preferably, defective individuals, from whom are largely recruited the neurotic, the insane, and the criminal classes.

The marks of this type are called the *stigmata of degeneracy*, and may be divided into the morphological and the functional.

Morphological Stigmata.—Commencing at the crown of the head, the *whorl of hair* at the vertex which normally is close to the median line may be widely displaced or duplicated. The *cranial conformation* is often abnormal in outline, capacity, or dimensions. The occipital protuberance and ridges, the frontal eminences, and the mastoid processes are usually excessively developed. The *facial angle* is reduced, the contour of the *face* asymmetrical, the *lower jaw* disproportionately large and prognathic. The *hard palate* is sharply vaulted, the *dental arcades* narrow, disproportionate, saddle-shaped, or angular and badly articulated; the *teeth* defective, misplaced, with sometimes persistence of milk-teeth late in life. The *ears* are disproportionate in size, misplaced, malformed, particularly at the root of the helix, which may bifurcate, or the tragus and antitragus are misplaced, while the concha is crumpled or has a tendency to stand out widely from the head. The *eyes* show notable defects, extreme refraction anomalies, squints, different colored irides, and disproportionate lids and palpebral openings. *Deviation of the nose, septal deformities, harelip, cleft palate,* and remnants of *branchial clefts* in the neck or in front of the ears, and the presence of other teratological deficiencies are frequent in this class of persons.

On the part of the trunk, *spina bifida, sacral growths of hair, deep sternal furrows and concavities, or disproportion between thorax and abdomen* are to be noted.

The Extremities.—The upper and lower limbs may be disproportioned to each other or to the trunk. They may be mismated in length and development. The *hands* and *feet* may be too small or too large. There is often a tendency to left-handedness and left-sided overdevelopment. *Deformities of the fingers,* such as syndactyly, polydactyly, deviations, distortions, excessive length or shortness, especially undersize of the ring and little fingers as compared with the rest of the hand, are common in degenerates.

The *genitalia* in the male, besides a general lack of growth, are frequently developmentally defective, presenting hypospadias, epispadias, extrophy of the bladder, cryptorchidism, congenital phimosis, scrotal fissure, etc.; while in the female, imperforate hymen, double vagina and uterus, and hypertrophied clitoris and labia are not rare.

Taken *as a whole*, the degenerate physique is often marked by a diminished stature and an inferior vigor. Many neurotic males present the general body conformation of the opposite sex, including sloping, narrow shoulders, wide hips, excessive pectoral and pubic adipose deposits, with a lack of masculine hirsute and muscular marking. The female may present masculine characteristics, and in each case the opposite sexuality may be further manifest in the actions, dress, manners, voice, and mental qualities of the individual. Both sexes may retain the physical attributes of childhood,—*infantilism*,—and in these cases the mental development is always retarded.

On the part of *the skin*, albinism, melanism, and multiple nevi are sometimes degenerate accompaniments. A general lack of thorough development in the dermal structures is manifested by defective hair and nails and simplicity in the papillary lines of the finger-tips.

The functional stigmata of degeneracy show themselves: (1) *Mentally*, in defective mind qualities. These vary in degree from idiocy to simple retardation of speech development, in aberrant mental and moral tendencies, among which may be enumerated destructiveness, wilfulness, indecency, deceit, and sometimes extreme acuteness and even precocity in limited fields. Genius is essentially abnormal however valuable it may be to the individual and to the race. It is often attended by many of the physical stigmata of defect.

(2) *Physically*, may be mentioned backwardness in walking, stammering, incontinence of urine, merycism, color-blindness, deaf-mutism, perverted tastes, and cravings leading to alcoholism and other stimulant addictions. Perversions of the genesic sense, marked by sexual crimes and debasing practices, are also common. Degenerates have frequently a lack of adaptability to their environment, and so more or less strongly depart from the type and tend to extinction, subjugated by the law of survival of the fittest.

In estimating the various marks of degeneracy it is clear that very few of them, taken alone, would justify the classification of their possessor among the defectives, and it is true that a very great many of the minor stigmata may be present in a given case, associated with strong mental, moral, and physical attributes. All of them, from cleft palate to moral imbecility, are referable to defective development. However, in the presence of numerous indications of physical defect we are entitled to expect the association of their mental and neural analogues. Hence their importance to the neurologist.

The mental condition of the patient should not be overlooked. Disturbances in the psychic sphere are very common in nervous disorders and often overshadow them. Persistent *depression* or *excitement* out of proportion to their causes, and *delusions* and *hallucinations* that may or may not be properly recognized and corrected by the patient, require close scrutiny. Especially in hysteria are we confronted by a train of mental symptoms, attitudes, and reactions that may easily be confounded with insanity or which actually carry the patient over the rather broad, dividing neutral ground into the realm of alienism. *Loss of self-control, irritability, increased emotionalism,* and vague or formulated *apprehensions* are the ordinary concomitants of neurasthenia. Many cerebral diseases produce *unconsciousness.*

Sleep in nervous patients is one of the most important practical considerations. If it is disturbed, seek the cause, remembering that habits of wakefulness are easily formed. Distressing, formulated, and repeated *dreams and nightmares* are the neurasthenic's portion and the expression of his lowered nervous and physical tone. *Somnambulism,* nocturnal urinary *incontinence* and *night-terrors* are the common property of nervous individuals in childhood. Some patients find difficulty in falling asleep, others in remaining asleep; others are simply unrefreshed upon awaking. The selection of soporific remedies and the time of their exhibition turn upon such considerations.

Memory.—Nearly every nervous invalid asserts a *loss of memory,* which rarely, however, exists. This mental faculty varies not only greatly in individuals, but is subject to great modifications in a given individual under different conditions of health and age. To the keen perceptions of a child everything is novel, is deeply imprinted in the mind, and is rarely forgotten. Later in life a new face or name is no rarity, is not sharply apprehended, and its recollection is consequently difficult or impossible. The old, in part for this reason, remember their early experiences better than more recent happenings. In physical ill-health and in conditions of mental abstraction or introspection, as in hypochondria, hysteria, and neurasthenia, the alleged loss is really a lack of the mental concentration that constitutes the essential basis for good memory. In such cases this may be demonstrated by a few questions on remote personal happenings, which will usually be recited with extreme minuteness and detail.

CHAPTER II.

THE GENERAL PHYSICAL EXAMINATION.

Present Condition.—What has gone before prepares the way for a thorough physical examination. Whenever possible, the clothing of the patient should be entirely removed, as study of the physical human outlines is most valuable. Without this step spinal deviations, chest deformities, lack of symmetry in the limbs, or other serious defects of the most important diagnostic character may escape notice. Upon sketch outlines of the human figure supplied in text-books and by dealers abnormalities of form and function may be indicated with precision. No lesion is too slight to be overlooked, and no assertion of functional health is to go unquestioned. Remote conditions are not infrequently causal of central disturbance, and central mischief is manifested by peripheral states. The nutritive process may first engage attention.

The Alimentary Tract.—The condition of the *teeth* in relation to mastication and abnormalities of position or evidence of inherited syphilis, the color of the *gums* with reference to anemia or evidences of metallic poisoning, such as the blue line of lead and the sponginess of mercury and phosphorus, can be noted at a glance. Particular attention is to be directed to *the tongue.* Aside from indicating the state of the stomach, it may give important evidence of nervous diseases. The fine fibrillar twitching of general paresis and bulbar palsy, the tremor of alcoholism, the contortions of chorea, the lack of motility and atrophy in labioglossolaryngeal paralysis, and the deviation on protrusion in hemiplegia are positive signs of great value. Abnormalities of *appetite* for food, gastric and intestinal *indigestion*, *constipation*, and *rectal conditions* are significant in many ways. Attacks of *colic*, *vomiting*, *diarrhea*, and rectal *tenesmus* have special bearing on the condition of the reflex spinal centers. The condition of *spleen* and *liver*, as in acute and malarial infections and chronic alcoholism, may give important indications of constitutional and local states that have a relation to the nervous phenomena under investigation.

The Respiratory Organs.—In the *nasal* and *pharyngeal spaces* inflammations, new growths, or irritation zones may furnish the starting-point for neurotic states of the most varying nature, as hysterical sneezing, spasmodic asthma, and pronounced neurasthenia. *Laryngeal palsies* and *spasms* require a careful topical examination. In the condition of the *lungs* and *pleuræ* we seek for local explanation of various symptoms, such as respiratory pain and oppression, costal neuralgia, continuous cough, or for the evidence of tubercular deposits, explanatory, perchance, of a cachexia that might otherwise be erroneously referred to nervous depression.

Circulatory Apparatus.—The condition of the *heart* is revealed only by a thorough physical examination of its position, size, action, and valvular competency. The condition of the *arteries*, patent to the eye in a tortuous temporal, to the finger in radial atheroma, should be still more extensively investigated in the femoral, brachial, carotid, and other superficial regions. The condition of the *blood-pressure*, as shown by arterial tension, on the two sides of the body, near the heart, and at a distance, is worthy of careful study. It enables one to draw analogical conclusions as to the circulatory apparatus of the central nerve-organs. The condition of the arteries is the best index of the real age of an individual. In them we often find evidence of a premature decay out of all relation to the years that have been lived. Sphygmographic tracings, as a method of record and precision, have their own value. The *pulse*, by its lack of rhythm, particularly by a tendency to great variation in its rate, depending upon slight exciting causes, often shows the unstable nerve-tone of the patient, or a general asthenia. *Flushings*, *mottlings*, *local anemias* and *edemas* are vivid expressions of angioneurotic disturbances. The *blood* must be examined for parasites, hemoglobin, and corpuscular conditions. The severe anemias have a very important relation to brain and spinal symptoms. The presence of cholin in the blood is frequent in diseases marked by degeneration of nervous tissues.

The temperature may be greatly modified by nervous diseases. Organic brain-lesions may upset the balance between the thermotaxic and thermogenic centers, producing either a very high or a markedly subnormal body-heat. In hysteria a very high range of temperature is sometimes noted without the usual concomitants of fever. In cerebral hemorrhage and basilar meningitis the temperature is often below the normal. Early in cerebral hemorrhage the paralyzed side presents usually a disproportionate elevation of a degree or more of heat over the opposite half of the body, as is shown by axillary temperatures. The temperature of the paralyzed side later becomes subnormal. Slight variations of the central normal temperature, usually in an upward direction, are frequently observed in pure neurasthenic states, while the extremities are commonly cold.

The Integument.—From the appearance and condition of the cutaneous expanse much is to be learned as to the general health of the individual and the activity of his physical functions. The skin may be greatly modified by nervous maladies. In some instances the dermal manifestations make up the major part of the disease, or the dermatosis may be an associated feature of other neurotic disturbance. All varieties of *urticaria* are of frequent occurrence among the neurotic. *Dermographia* and the *tache cérébrale* of meningitis demonstrate the vasomotor irregularities. *Herpes* and *morphea*, limited to the anatomical distribution of nerves or spinal segments, as in zoster on the face, trunk, or limbs, declare the nervous involvement. *Neuralgias* of long standing are frequently marked by dermal changes of increased or decreased nutrition, as witness the thickening of the skin of the face in neuralgia of the fifth cranial nerve and the subsequent blanching or the actual loss of the eyebrow and hair. The neuritides, if of a severe grade, show dermal dystrophy as well as muscular wasting. The epithelial structures involved may take on increased growth if vascular stasis favor increased nutrition, giving rise to scaliness of the skin and increased growth of the hair and nails. More frequently atrophic changes follow; the skin is thinned and glazed, the epithelium scant and poorly protective of the more highly organized subjacent tissue. The hair becomes dry, brittle, and sparse, and the nails rough-ridged and sometimes covered with overlapping scales. *Pigmentary changes, swelling*, and *blue edema* are not infrequent in hysteria. The enormous *thickenings* in myxedema and acromegalia are also due to perverted trophic control.

Genito-urinary Tract.—In the genito-urinary tract are found many conditions bearing an intimate relation, both causal and symptomatic, to nervous diseases. Some of them are overlooked or unknown to the patient, and others receive altogether too much attention at his hands. A thorough clinical examination of *the urine*, which should be quantitative as well as merely qualitative, is best made from a sample of a carefully measured twenty-four-hour collection. It shows at once the eliminative powers of the organism through the important excretion outlet of the kidneys. A lessened output of *urea*, or the presence of *albumin* or *sugar*, give important data as to the blood-state and may explain grave cerebral manifestations, such as convulsions and coma. A very *low specific gravity* is noted after hysterical attacks. A large quantity of *phosphates* and *oxalates* is common to many neurasthenic conditions, and an *ammoniacal urine* is usual in paretic states of the detrusor urinæ. After an epileptic attack the specific gravity and *solid constituents* of the urine are increased.

The microscope, besides giving evidence of organic disease, such as nephritis, pyelitis, and cystitis, may show *spermatozoa* from a relaxed control of the outlets of the seminal vesicles, but more often demonstrates that the deposit considered seminal by the patient is devoid of testicular products.

Anuria in nephritis is of most serious import, though it may exist almost indefinitely in hysteria when associated with persistent emesis, whereby the uric products are vicariously ejected.

The state of the *kidneys* is made out largely by the investigation of the urinary secretion, but the *bladder* and *ureters* are open to more proximate methods. The size, expulsive and retentive powers of the bladder, its contents, and the condition of its mucous lining are, in suitable cases, to be investigated with precision. Loss of *sphincteric control* of the bladder in paraplegic and ataxic conditions is usual, while in meningitis, and in comatose states generally, *retention* of urine is to be expected and provided for.

The *external genitals* rarely give much information. In males *preputial adhesions* and accumulations or a long *phimotic prepuce* may be the source of irritation and the inciting cause of general nervous phenomena. Here, as elsewhere, any abnormal and correctable state should not escape appropriate attention. This is emphatically true of the *deeper-lying generative* organs in the female. Undoubtedly undue importance has attached to them and much ill-advised meddling has been bestowed, but a lack of integrity on their part should certainly engage methodical treatment.

The *genesic sense* is usually blunted or completely destroyed in advanced locomotor ataxia and spinal lesions which cut off peripheral sensation. It is reduced in all depressed physical states, whether associated with marked nervous phenomena or not. On the other hand, the genital reflex may be accentuated in lateral sclerosis of the cord, and priapism, unattended by increased desire, may be a troublesome feature. In injuries to the cervical portion of the cord, priapism is likewise common.

The question of sexual irritation and overindulgence calls for more than ordinary thought. Excess is a matter purely relative to the individual and his condition at the time of indulgence. Overuse of any organic function is shown by persistent fatigue and irritable prostration. This may here furnish us a working criterion, but we are to remember that matters have already gone too far when the great margin of natural reserve power has been overdrawn and even temporary debility produced. Thus, an amount of masturbation or sexual indulgence insignificant in a sturdy individual, is sufficient in the defective neurotic to induce a profound depression.

touching the tip of his nose, the lobe of his ear, or the finger of the examiner. In the lower extremity we ask him to touch a given object with the point of the foot or reproduce with the other the position given a limb by the examiner. In making this last test it is necessary to avoid furnishing the patient such information as might come from contact with the bed or personal clothing. Standing with the eyes closed and the feet together, reducing the base of support, is often attended, in spinal and other nervous diseases, with swaying of the body and a tendency to fall. This constitutes *static ataxia*, and is known as the Romberg symptom. It may be graphically recorded by attaching a marking-point to the patient's head, which traces his ataxic movements on a prepared surface held at the proper level. Having the patient walk backward with closed eyes or stand on one foot increases the ataxia.

Incoördination may be very great, so that the blindfolded patient does not come within several inches of his nose in his attempts to touch it with his finger, and has no certain knowledge of the position of his limbs under the bed-covering except as he informs himself by sight. While standing, he may fall at once if his eyes are closed, or even reel in his chair.

The following example of ataxic writing shows incoördination very clearly, and indicates another important test of muscular balance :

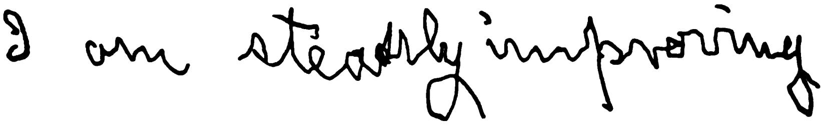

Fig. 2.—A specimen of ataxic handwriting.

Increased Motility.—Perhaps motility in nervous disorders is more frequently excessive than deficient—that is, beyond the purposes of the patient. Paretic conditions, too, are not infrequently associated with irregular and involuntary muscular movements.

Tremors are the accompaniment of overexertion and emotion in health, but often become significant symptoms of nervous disturbance and disease. A tremor that exceeds six to the second is called *rapid*, one below that rate *slow* or *moderate*, while the amplitude of the muscular vibration or excursion is indicated by the adjectives *fine* and *coarse*. Tremors occur sometimes during complete rest, as in paralysis agitans, or only when the involved muscles are called upon to act—the "*volitional*" or "*intention*" tremor, as in multiple sclerosis. While tremors may involve the entire muscular system, as is often the case in exophthalmic goiter, they may be restricted to a single limb or group of muscles, to a single muscle, or even to a few muscle fibrils. This last constitutes a *fibrillary tremor*, or, rather, fibrillary spasm, because it is usually devoid of rhythm, and presents only one or a few irregular twitchings under the integument. They may sometimes be provoked by stroking or tapping the surface or putting the muscle into operation.

lifting power of the back, the legs, or the arms is readily ascertained. Readings on such an instrument are of the greatest value for clinical records and as a means of comparison at future times.

But without any such instrument the physician can roughly test every group of muscles by opposing the efforts of the patient. To examine flexor and extensor muscles of the wrist, elbow, and shoulder, instruct the patient to resist your efforts to communicate motion to the respective joints. The same can be done in the lower extremity with the ankle and the knee. The hip is tested by having the patient mount a chair, ascend stairs, and raise the limb to a horizontal position. Both hands of the examiner grasped by those of the patient as vigorously as possible enables the physician to compare their strength.

In hysterics and malingerers it is sometimes difficult to feel that full efforts have been put forth, and in other instances muscular exertion is inhibited by pain or tenderness. In these cases particularly, the muscular tests may be advantageously varied and repeated by securing a large number of movements, such as having the patient stand on one foot, rise on the toes, stoop, crouch, lift chairs, and execute other tasks when he is not conscious of the purpose in view. Small movements of the fingers are very clearly studied as the patient buttons his clothing or handles different objects, which may, with design, be placed in his way.

Where the degree of feebleness is slight, it perhaps is manifest simply as fatigue after exertion or in walking a comparatively short distance. In some cases muscles which at first register considerable strength are rapidly exhausted by a few full contractions. In others initial efforts are weak, but quickly increase to a normal power, making it needful to always take an average of several tests.

The *limits of the paralysis* or paresis must be precisely noted, whether confined to a single muscle or muscle group, to the distribution of a single nerve or group of nerves, to a single limb, to the face, to one side of the body or to more. If the involvement is wide-spread, it will probably vary in degree in different regions, and this is also of importance. The terms *monoplegia*, *hemiplegia*, *paraplegia*, and *diplegia* are employed respectively as the face or a single extremity, the lateral body-half, both lower extremities, or both sides of the body are involved.

Not only is it necessary to record the strength of the muscles under examination, but also the manner in which they perform. For all precise movements, complete balance of muscular action—*synergy*—is a prerequisite. If the flexors of a joint are weakened, the extensors not only fail to execute their function with exactness, but their strength is also diminished and the resulting volitional movement is weak or clumsy in consequence. In conditions marked by errors of sensation, especially by impairment of the muscular sense which gives a knowledge of the position of our members and enables us to estimate weight, movements become uncertain. This uncertainty is generally exaggerated if the movements are not guided by the eye. *Incoördination* of movement results. This is sought for, and, if present, is demonstrated by having the patient, with closed eyes, touch given points, either on his own person or elsewhere, as by bringing the index tips of both hands together, touching the tip of his nose, the lobe of his ear, or the finger of the examiner. In the lower extremity we ask him to touch a given object with the point of the foot or reproduce with the other the position given a limb by the examiner. In making this last test it is necessary to avoid furnishing the patient such information as might come from contact with the bed or personal clothing. Standing with the eyes closed and the feet together, reducing the base of support, is often attended, in spinal and other nervous diseases, with swaying of the body and a tendency to fall. This constitutes *static ataxia*, and is known as the Romberg symptom. It may be graphically recorded by attaching a marking-point to the patient's head, which traces his ataxic movements on a prepared surface held at the proper level. Having the patient walk backward with closed eyes or stand on one foot increases the ataxia.

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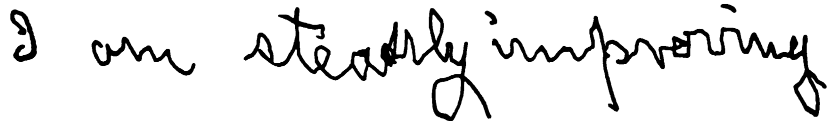

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Eshner,[1] as a result of an extended series of experiments, concludes: (1) All muscular movements are made up of a series of elementary contractions and relaxations, which may be appreciable as tremors in conditions of both health and disease. (2) The differences between different tremors are of degree rather than of kind—*i. e.*, no form of tremor is distinctive of any one disease or group of diseases. (3) No definite relation exists between one form of tremor and any other. (4) The frequency of movement is in inverse ratio to the amplitude and vice versa. (5) Habitual movements are performed with greater freedom from tremor than unusual movements. (6) There is no material difference between the movements of the two sides of the body, except as related to proposition 5.

In *testing for tremor* the patient is directed to stretch out the arms with the fingers extended and separated as widely as possible. The difficulty becomes at once apparent, or is felt as a thrill by the examiner's hand grasping the wrist or placed against the finger-tips of the patient. Care must be exercised not to mistake the origin of the tremor, as vibrations of the head and extremities may be *communicated* from a distant point. Again, tremor in the tongue and face is best shown when the patient, with closed eyes, protrudes the tongue vigorously for a few moments. Intention tremors are usually best shown when the patient attempts to carry a full glass of water to the lips, to write with a pen, or to execute other delicate coördinate movements. By means of registering apparatus a tracing of the tremor may be obtained in a graphic manner, and for this purpose the sphygmograph may sometimes be employed. The following specimen of writing in a case of paralysis agitans most clearly shows the amplitude, rhythm, and, by timing the effort, the rate of the tremor:

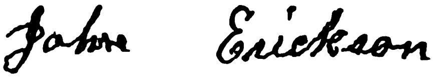

Fig. 3.—Specimen of handwriting showing tremor of paralysis agitans.

Spasms.—When a muscle or group of muscles contracts more or less energetically without the conscious intention of the individual, it constitutes a *spasm*, and is *clonic* or *tonic* as it is frequently repeated or steadily continuous. A painful spasm is usually called a *cramp*, though a facial spasm, accompanied with pain, receives the now classical designation of *tic douloureux.*

Long-continued spasm leads to *contracture*, a condition in which the muscle eventually becomes structurally shortened. Contractures follow, also, through natural tonus, when a muscle is for a long time unopposed by its physiological antagonist. *Postplegic movements* sometimes follow brain-lesions of long standing. The paralytic portions of the body manifest peculiar involuntary movements, which may vary from slight twitchings of a choreic character to the never-ceasing, purposeless activity shown in *athetosis*, where the fingers and toes work in a tentacle-like, or ameboid, manner. Voluntary efforts usually increase these athetoid movements. In hemiplegia, at times the paralyzed members duplicate the voluntary motions of the sound side, producing *associated movements.* They are particularly likely to occur under instinctive action, such as stretching and yawning, or under strong voluntary efforts with the sound side, as in the attempt of a right-handed, right-sided paralytic to write with the left hand.

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[1] "Jour. Exper. Med.," 1897.

Convulsions consist of more or less widely-distributed, persisting or repeated spasms, associated usually with disturbed or abolished consciousness. They are symptomatic of numerous cerebral disorders, injuries, and intoxications. We speak of them as being *general* when the entire body is affected, or *local* when the convulsive movements are confined to a limited portion of the muscular system, as the face, hand, or lower extremity. Local convulsions are frequently styled *Jacksonian,* especially if marked by progressive extension to adjacent muscles, with tardy, slight, or no involvement of consciousness. The initial spasm of such fits is called the *signal symptom,* indicating fairly the cerebral center in which the muscular storm arises. Convulsions are *tonic* or *clonic,* as the spasms of which they are made up are long maintained or frequently repeated.

The careful investigation of a fit, and the importance attaching to its details, their order of appearance with all associated precursory and sequential phenomena, have already been touched upon. When these clinical facts must be learned from lay persons even of the most intelligent character, the difficulties are often insurmountable. In some cases a trained medical man or a well-instructed nurse must remain with the patient for the purpose of getting a complete description of the attack.

In rare cases the ordinary muscular tone is abnormally increased, —*myotonia.* This gives rise to a condition analogous to a tonic spasm, and voluntary effort is thereby delayed, as in Thomsen's disease.

Reflexes.—In the muscular reflexes we have a series of signs which give information regarding both the spinal centers, and the nerve-paths above and below these centers. They have great diagnostic value and localizing importance. A knowledge of the spinal-cord segments to which they belong, and of the anatomical relation of these segments to the vertebral bodies and spinous processes, with the association of muscle and sensory areas, forms the basis of spinal localization.

A muscular reflex action is the result of a peripheral stimulation reaching motor spinal centers and thence centrifugally manifest in a contraction. Thus, in the pupil, the stimulation caused by light falling on the retina travels by the sensory limb of the reflex arc to the medullary center, and there calls forth energy which flows down the motor limb and causes pupillary contraction. A blow on the patellar tendon stimulates centripetally the lumbar center, and a contraction in the extensor muscles of the leg results in the "knee-jerk." The normal activity of the reflexes requires not only the integrity of the center and both limbs of the reflex arc, but a proper association of the center with the higher-lying cerebral levels. Any block or interruption in either limb of the arc, or the destruction of the spinal center, abolishes the reflex. At the same time it is a general rule that pathological conditions interfering with the free communication between spinal center and cerebral cortex tend to increase the reflex manifestations of the cord. In man, however, the absolute destruction of all such communication—in other words, complete division of the cord—produces abolition of muscle reflexes below the level of the lesion. Sensitiveness in the part tested is ordinarily attended by an increase of reflex irritability, as, for instance, in rheumatism.

Reflexes have been variously described as skin, superficial, deep, tendon, muscular, and organic,—distinctions that have no real value, as there is no essential physiological or anatomical difference. Many of them can be inhibited voluntarily by the patient, and all require complete passivity on his part for their proper investigation. Some only appear as a result of disease. As a rule, the muscle under examination must be slightly stretched, and then the blow or passive motion, which suddenly increases its tension, gives rise to the reflex movement.

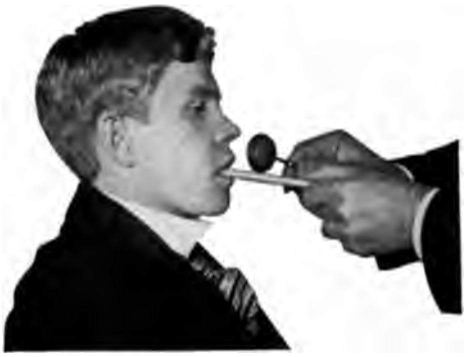

Fig. 4.—Method of eliciting the jaw-jerk.

The contact or threatened contact of any object with the eyelids or conjunctivæ causes a *palpebral reflex*—the closing of the eye.

A *supra-orbital* reflex has been described by McCarthy. It is elicited by percussing over the supra-orbital nerve either at the point where it emerges or somewhat higher up in its field, and consists of a fibrillary twitching of the orbicularis palpebrarum. It should not be confounded with the palpebral reflex. It is absent in severe injury or disease of the facial and of the fifth cranial nerves.[1]

The *pupillary reflex* is manifest under several conditions: (1) If the eyes have been closed for a few moments and then suddenly opened in a strong light, or if in a dark room a beam of light is thrown upon them, the dilated pupils quickly contract. (2) The normal eye shows a decided pupillary contraction if focused on a near object—say, at eight inches—after being directed at a distance of over twenty feet. This latter is sometimes called the *ciliary reflex*. These normal reactions are variously modified by disease. They may be simply sluggish, they may be entirely absent, or they may be dissociated. In locomotor ataxia and in paretic dementia, when the posterior portions of the cervical cord are sclerosed, the pupils usually are narrowly contracted. They then respond to efforts of visual accommodation only, and are quite insensitive to light. This is known as the *Argyll-Robertson pupil*. Rarely the opposite condition has been noted in other diseases. The action of certain drugs on the pupil, as opium and belladonna, is always to be kept in mind when looking for this reflex, and the eyes must be separately observed, as only one may be affected. The patient must not be allowed to close the eyes vigorously in testing for the pupillary reflex to light, as this act causes the pupils to dilate, though covered by the lids. In certain conditions, when a portion, usually one-half, of the retina is blind or insensitive, a narrow ray of light made to fall upon this part of the optic nerve, by being thrown obliquely through the pupil, causes mydriatic contraction. Wernicke first pointed out that this occurred only when the lesion causing hemianopsia was situated back of the geniculate bodies in the optic radiation or in the visual centers, and the condition is known as *Wernicke's sign*. (3) Sharp pinching of the skin on the side of the neck and severe pain in general cause a dilatation of the pupil.

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[1] "Neurol. Centralbl.," Sept. 1, 1901.

The *jaw-jerk*, or *mandibular reflex*, is produced best by placing a ruler or similar object on the lower incisor teeth while the mouth is partially opened, and striking down upon it with a percussion hammer. This is followed by a quick contraction of masseter and temporal muscles, and usually by the active closing of both eyes.

The *pharyngeal reflex* is the spasmodic action, familiar to every one, produced by tickling or stroking with any object the faucial pillars or the walls of the pharynx, and sometimes even the soft palate or base of the tongue. It is commonly absent in hysteria. Patients who have been subjected to much local pharyngeal treatment sometimes acquire much tolerance of manipulation and control this reflex completely.

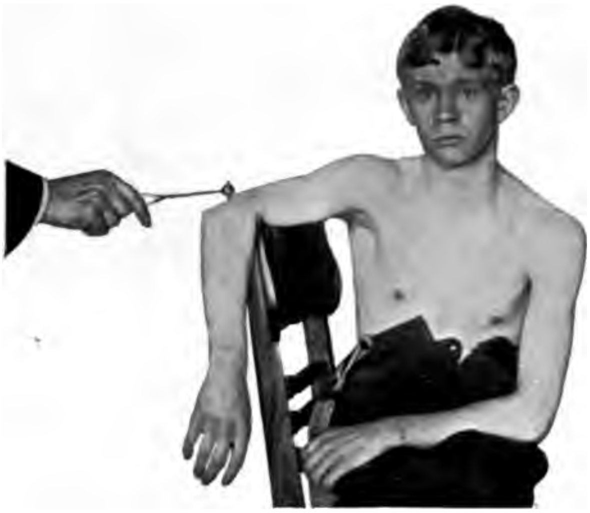

Fig. 5.—Method of eliciting the elbow-jerk.

In the *upper extremities* there are a large number of muscle reflexes which may be elicited by striking on the insertion tendon after so placing the member that the muscle under investigation is put in moderate passive tension. Unless the patient completely relaxes, it may be quite impossible to demonstrate the reflex, and it is often difficult to secure this relaxation even in well-intentioned persons, unless their attention is distracted.

The various portions of the *deltoids*, *pectoralis major*, *trapezius*, *serratus magnus*, and *scapular muscles* may be made to react in this way under the percussion hammer in most persons, but are much more active in neurotics and in those spinal diseases marked by increased reflexes. With the arm extended and supinated, a tap at the insertion of the *biceps* causes a flexion movement at the elbow. The *flexors of the wrist and fingers* similarly respond while the extremity is in this position.

With the elbow flexed, as by allowing the arm to hang over the back of a chair, the *triceps* is actuated by a tap above the point of the elbow. With the forearm slightly pronated and the hand hanging, as in the usual position on a chair-arm, the *wrist* and *finger extensors* can be similarly examined. Of the small muscles of the hand, only the *palmaris brevis* can readily be brought into play in health. This is done by making pressure over the pisiform bone and lower end of the ulna with the thumb and forefinger, and causes a grooving in the ulnar border of the hand just above this point.

In some spastic cases the passive sharp flexing of fingers and wrist may develop a *wrist clonus,* consisting of rapidly repeated movements of extension and flexion at the joint, which tend to persist as long as gentle tension is maintained by the examiner upon the extensors.

On the *dorsum of the trunk* there are a series of reflexes which, below the scapulæ, are not of much diagnostic value, and which can usually be demonstrated by stroking, pinching, or, preferably, percussing the muscular masses. *Anteriorly,* with the patient lying supine and the abdominal wall relaxed, a tap on the costal cartilages on either side of the xiphoid depression causes a dimpling or lateral movement at this point, called the *epigastric reflex.* A similar tap on the costal border in the nipple line, acting through the abdominal oblique muscle, produces the *abdominal reflex,* most noticeable at the umbilicus, which is promptly drawn toward the side percussed and in the oblique direction indicated.

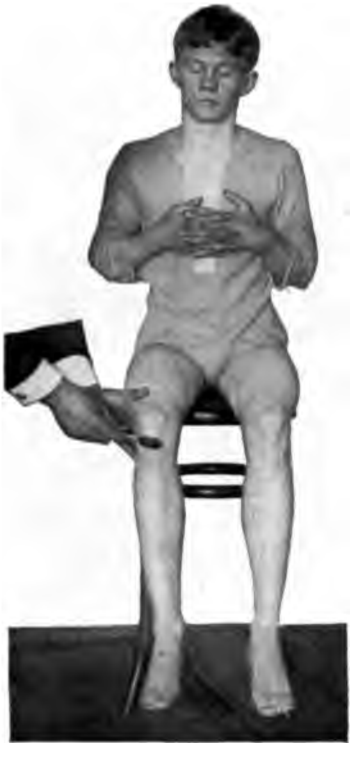

Fig. 6.—Method of eliciting the knee-jerk and reinforcing it by Jendrassik's method.

In the *lower extremities* we find that a series of taps along the origin of the *great gluteal muscle,* when the patient is erect or prone, are followed by contractions in corresponding segments of that muscle, and a tap near the anterior superior spinous process starts the *tensor faciæ latæ femoris.* The *knee-jerk,* or *patellar reflex,* being easily examined and frequently modified by disease, is one of the most important of the muscle reflexes. It is elicited generally by having the patient cross one knee over the other while sitting. The under limb, with the knee at a right angle, should support the upper, which gently rests over it with all muscles relaxed. A *smart, quick blow* with an object of some ounces' weight, as with the back of a thin book or the ulnar border of the hand, upon the patellar ligament or just above the patella, is followed by a contraction of the anterior thigh-muscles, causing the suspended foot to move forward an inch or two. If the patient is in bed, the limb to be examined may be lightly placed over its extended fellow, crossing it at the knee, and then the blow employed, or with the patient on his side, the knees partly flexed, the same thing may be done. When difficulty is encountered in securing this response, it is to be remembered that unless the muscles are fully relaxed the patient may inhibit the phenomenon, or that it may be so slight as to escape attention. If the patient be placed on a high chair or on the edge of a table so that the legs are pendent, and at the same time his attention be diverted, the jerk may usually at once be shown. It can also be *reinforced*, as described by Jendrassik, by having the patient grasp some object vigorously with his hands, or by merely clenching his hands at the time the tendon is struck. It should only be considered abolished when, thus reinforced, with the limbs unclothed, the eyes closed, and the unemployed hand of the examiner upon the rectus femoris, no response can be detected. Similar plans of reinforcement are of value in testing other reflexes, and serve to divert the patient's attention.

In some cases, where the reflex activity is pronounced, by pushing the patella sharply downward when the limb is extended a *rectus reflex* is produced. If, upon continued downward pressure, a number of rhythmic contractions ensue and are disposed to continue, we have the *rectus clonus*. Taps over the insertions of the adductor group and over the tendons of the knee flexors, in cases marked by increased reflexes, produce corresponding muscular contractions.

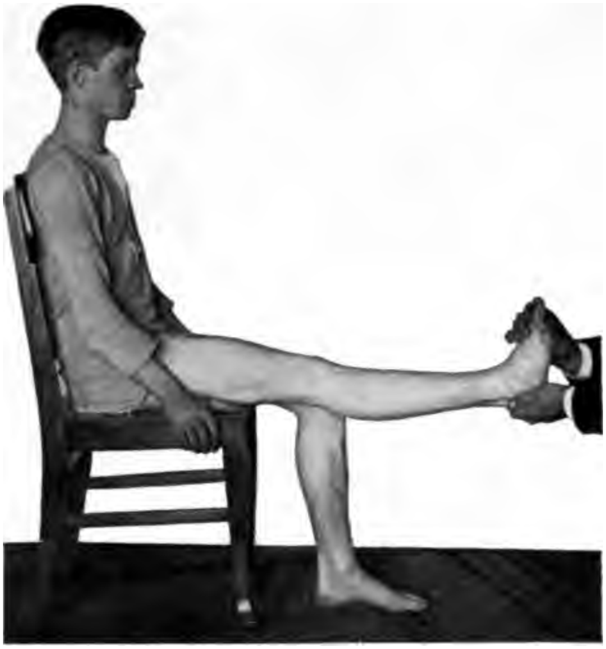

Fig. 7.—Method of eliciting ankle-clonus.

In conditions of *reflex* or *myotatic irritability*, if the knee be extended and the foot strongly flexed dorsally, a sharp tap on the upper and outer portion of the leg, over the extensors of the foot, causes a contraction of the calf-muscles, and this response is called the *front-tap contraction*. With the foot in the same position, a tap upon the *Achilles tendon* causes an extension of the ankle-joint. If there is doubt about the presence of the *heel-tendon reflex*, place the patient on a chair in the kneeling posture, direct him to grasp the chair-back firmly, make slight pressure over the ball of the foot to extend the tendon and use the percussor. The early absence of this reflex in sciatica and locomotor ataxia renders its examination important. The *peroneal muscles* likewise respond when properly percussed, the foot being first turned somewhat inward to put them on the stretch.

Ankle-clonus or *foot-clonus* is tested by sharply flexing the foot dorsally with the knee extended, and consists in rhythmic movements of the foot upon the leg, caused by repeated contractions of the calf-muscles. They persist usually as long as pressure is maintained against the ball of the foot. Sometimes it can best be developed by having the patient, as he sits, place the foot beside the chair in such a way that the weight of the limb is supported by the point of the foot. The clonus movement then causes the extremity to dance.

A *paradoxical* persistent contraction is occasionally found when the ends of a muscle are suddenly and passively approximated. For instance, when the foot is passively flexed on the leg, a tonic contraction of the anterior tibial muscles takes place. All other muscle reflexes are momentary, and occur under conditions of extension.

Tickling or stroking the plantar surface gives rise usually to well-known contractions of a wide-spread character, known as the *plantar reflex*. They may be confined to the flexors of the toes. Instead of flexion the toes may sharply extend, particularly the great toe, constituting the important *toe-sign of Babinski*, found only in disordered, usually degenerative, conditions of the pyramidal tracts. Oppenheim elicits this reflex by deeply and forcibly drawing the handle of the percussion hammer or the finger ends behind the inner border of the tibia in the lower third of the leg. Gordon,[1] of Philadelphia, has discovered also that very deep pressure of the calf muscles will in similar cases produce an extension movement of the toes.

When the skin on the inner side of the thigh, or when the scrotum is sharply stroked or pinched, the corresponding testicle is more or less actively drawn toward the pubic arch by the cremaster muscle. This *cremasteric reflex* must not be confounded with the slow contraction of the dartos tunic of the scrotum, of which it is entirely independent.

The sphincters of the bladder and bowel act reflexly upon the contact of any foreign body, and the destruction or serious impairment of these *sphincter reflexes* is attended by incontinence. The *sphincter reflex* of the bladder is tested by the introduction of a sound, noting the presence or absence of the sphincteric grasp. A finger inserted in the rectum distinctly feels the *anal sphincteric reflex*, if present. Pinching or pricking the skin about the anus causes an *anal reflex* consisting in a puckering of the mucocutaneous margin of the bowel outlet, and this may also be attended by a distinct sphincteric contraction.

When the corona glandis penis is pinched a contraction may be felt by the examiner in the perineum due to contractions in the bulbo-cavernosus muscles. This is called the *virile reflex*, and by some writers is thought to be an evidence of potentiæ coeundi.

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[1] "Jour. of Nervous and Mental Dis.," Feb., 1905.

In general, we may say: First, that the Argyll-Robertson phenomenon, Wernicke's sign, iridoplegia, ankle-, wrist-, jaw-, rectus-clonus, and Babinski's sign are never found in health, and are valuable objective signs of central disease. Second, that the abolition of the knee-jerk never occurs in healthy persons, and that the abolition of the sphincter reflexes is strongly indicative of central disease. Third, that the abolition of the pharyngeal and plantar reflexes, with increase of others, is presumptive of hysteria. Fourth, that a moderate intensification of all reflexes indicates neurasthenia. Fifth, that the abolition of all reflexes in a given anatomical area points to histological disease, either central or peripheral, in the arcs supplying those reflexes.

It should be pointed out that in hysteria and neurasthenia there is sometimes a *spurious ankle-clonus* that may be misleading, the foot making a few vibrations only upon the institution of the test. In this condition, too, there is a tendency for the reflexes to be wide-spread, so that a tap upon one patellar tendon may cause both legs to respond, start the upper extremities, or almost convulse the patient.

Myoidema, or idiopathic muscular spasm, is produced in certain irritable conditions by sharply striking across the muscle with a ruler or similar instrument, causing a local contraction at the point struck, with a bunching up of the muscular tissue that persists from a few seconds to several minutes.

CHAPTER IV.

TROPHIC CONDITIONS.

THE significance of abnormal variations in the nutritional conditions of a part is at once apparent when it is recalled that the growth and nourishment of all the structures of the body are presided over by trophic centers acting through peripheral nerves. For the proper nutrition of skin, muscle, nerve, and bone the integrity of the trophic center, of its peripheral path, and of its termination is essential. In other words, the anterior spinal cell and its polar prolongation in the efferent nerve, the lower neuron, can not be injured or destroyed without correspondingly impairing the function of nutrition in its area of distribution. All diseases, therefore, which affect the anterior spinal gray matter or the peripheral nerves are symptomatized by trophic changes in the associated parts. Further, in some local conditions of disturbed nutrition, where even upon minute examination of trophic cells and efferent nerves we can not discern any abnormality with all the means now at command, still we are justified by analogy in supposing some modification of this trophic energy, some dynamic change, some perversion of the function of the trophic apparatus relating especially to its center.

It is also evident that some substances act as stimulants to the trophic centers. This is seen in the hypertrophies of acromegalia and myxedema, where there is a perverted action of certain ductless glands. Other agents, as perhaps lead, may have a depressing effect upon these centers. The blood-supply of a part and its nutrition are intimately associated. The vasomotor and trophic apparatus are apparently mutually dependent, and as a matter of clinical fact it is constantly observed not only that in wasted tissues the blood-supply is greatly reduced, but that in hypertrophic states the vascularity is increased.

When the trophic apparatus of a limb is involved, the various tissues suffer in proportion as they are highly organized. Fibrous tissue, cuticular epithelium, and bone are but slightly modified, though their growth and repair may be stopped, while the more vascular and highly organized muscles and nerves promptly waste.

The Skin.—In conditions of acute *trophic irritation*, as in herpes zoster, the skin becomes injected, red, and painful, and the epidermis is raised in blebs or bullæ filled with serum. When the condition is one of chronic trophic irritation, the skin may become thickened, infiltrated, boggy, and show an actual hypertrophy of the subcutaneous areolar structure. The epidermis is increased in thickness, presenting scales and roughness, with an increase in the growth of hair and the secretion of sweat. The nails become rough, striated, coarse, and grow rapidly. *Diminished trophic energy* is marked by opposite conditions. The epidermis is thinned, imperfect, dry, and non-resisting to ordinary wear; the hair becomes brittle, scant, and sometimes turns gray; the nails grow slowly and imperfectly. The dermal envelope closely binds the underlying parts, loses its pliability and softness, and presents a reddish, purplish, or glistening whitish appearance.

The muscles show disturbance of trophic enervation very promptly. Acute poliomyelitis, or division of a peripheral nerve, is followed in a few hours by a muscular relaxation that is quite apparent, and in a few days the part looks wasted, though it may show no change on measurement. At the end of a fortnight a lessened size can be easily demonstrated. The sarcode elements rapidly alter and tend to disappear, finally leaving only the fibrous constituents to represent the muscle by a band of dense tissue, which shortens, gradually producing contractures and deformities.

In those conditions where the process is slower, the muscle does not change *en masse*, but is gradually invaded by the atrophy and shows some normal fibers very late in the disease and others that may be actually hypertrophied. In pseudohypertrophic paralysis the muscles become infiltrated with fat, which displaces the muscle elements, exaggerates the muscular firmness and outlines, and gives a false appearance of strength.

The peripheral nerves, when cut off from their trophic centers in the anterior spinal gray matter, show similar degeneration. Under conditions of trophic irritation they present proliferation of their fibrous structures and general thickening of their trunks, with more or less disintegration of their nobler elements. *Optic-nerve atrophy* is a visible instance of this kind.

The lowly organized, almost non-vascular **tendons and ligaments** in adult cases show little change when deprived of their nerve-supply, but even *bone* itself becomes less resistant, more fragile, and somewhat rarified under these conditions. In children bony growth is usually seriously checked.

The trophic condition of **joints** in nervous affections is one of great interest. In tabes dorsalis, and more rarely in other organic diseases of the brain and spinal cord, as in progressive spinal muscular atrophy and cerebral palsy, great changes take place in various joints. They become enlarged, enormously distended with fluid, and the bones disintegrate without any painful attending symptoms. This condition of *dystrophic arthropathy* was first clearly recognized by Charcot, and the articulations thus affected are often called *Charcot joints.* Again, arthritis of any character may be followed by extensive atrophy of the muscles above the joint, principally those of extension. This *arthritic muscular atrophy* in which the muscles waste, but conserve their electrical and reflex responses, is attributed to an irritant condition which arises in the inflamed joint. It affects the trophic spinal centers, and thence acts upon the nutrition of the particular muscles physiologically associated with that joint. It is positively known that division of the afferent path from the inflamed joint to the spinal center prevents the atrophic sequel.

Decubitus.—In acute processes involving the spinal cord an acute bedsore sometimes appears in a few days, completely denuding the sacrum. While this may be favored by pressure due to the position of the body and moisture from the urinary incontinence that is often present, it may also appear independently. Gross cerebral lesions are similarly marked in some cases by a sloughing of the buttocks, and this decubitus, as in the spinal lesions, is doubtless primarily due to interference with the trophic control, and in the case of hemiplegia affects the paralyzed side only.

Acute cystitis in paraplegic states, the low resistance of the tissues to pressure or heat, and the tardiness of repair have a similar significance and add greatly to the difficulty of caring for such patients.

Symmetrical limited deposits of fat and pigment, Raynaud's disease, symmetrical scleroderma, and scleroderma in the form of striæ atrophicæ following the nerves are clearly due to trophic disturbances, and are classed among the *trophic neuroses.*

Local conditions of trophic impairment may be a part of general ill-health or systemic diseases, and may in some cases be due to, but should not be confounded with, them. *Disuse* will also produce marked nutritional changes in a part, as inactivity does of the entire organism,—a fact to be borne in mind in relation to neurasthenics, hysterics, and malingerers.

CHAPTER V.

ELECTRICAL CONDITIONS.

NORMAL muscles and nerves respond to various methods of electrical stimulation in a precise and uniform manner. On the other hand, diseases which modify the electrical conductivity of nerves or their muscle-endings and diseases which modify the electrical stimulability of muscle-cells produce characteristic changes in their reactions when properly tested. These changes are of very great diagnostic importance. We require, for a reasonable examination of this nature, first, an induced or faradic current that can be increased from zero to a tetanizing strength; and, second, a constant or galvanic current of at least fifty volts' strength, that can also be gradually and uniformly increased or decreased at will or interrupted at pleasure. The first is supplied in many convenient and portable forms, of which, perhaps, the best is the chlorid of silver dry-cell battery, fitted with an induction apparatus of the Reymond type; the second by about thirty active Leclanché or equivalent cells, or the incandescent lighting circuit modified by a proper rheostat. For portable purposes batteries of various makers containing from twenty to fifty cells are available. Two electrodes are needed, one presenting from nine to twelve square inches of conducting surface, the other fitted with an interrupting handle by which the operator can easily control the current, and having a rounded conducting surface about one inch in diameter.

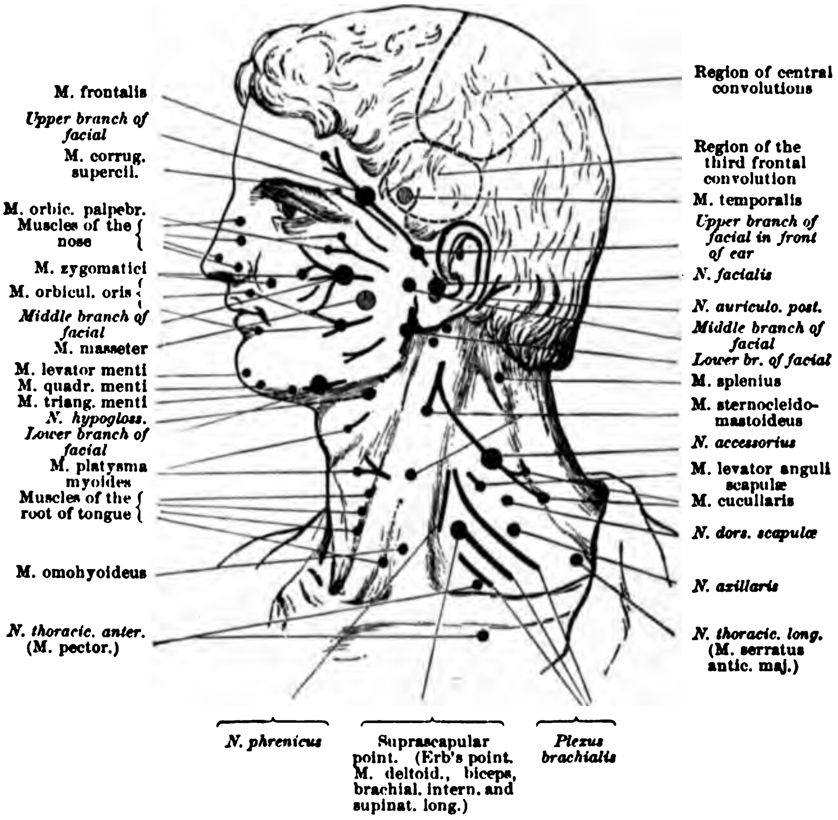

Fig. 8.—Nerves and motor points in face and neck.

When using the constant current, a milliamperemeter is of value and for purposes of record almost a necessity. Its readings show the amount of current in circuit at the particular time. It is to be remembered that all alterations in the condition of the test—such, for instance, as dryness of the electrodes or of the skin of the patient—modify the amount of electricity requisite to secure a given response. Probably identical conditions can not be secured even with the same patient twice in succession. All ampere measurement comparisons are therefore relative and approximate. Cells vary so much in activity at different times

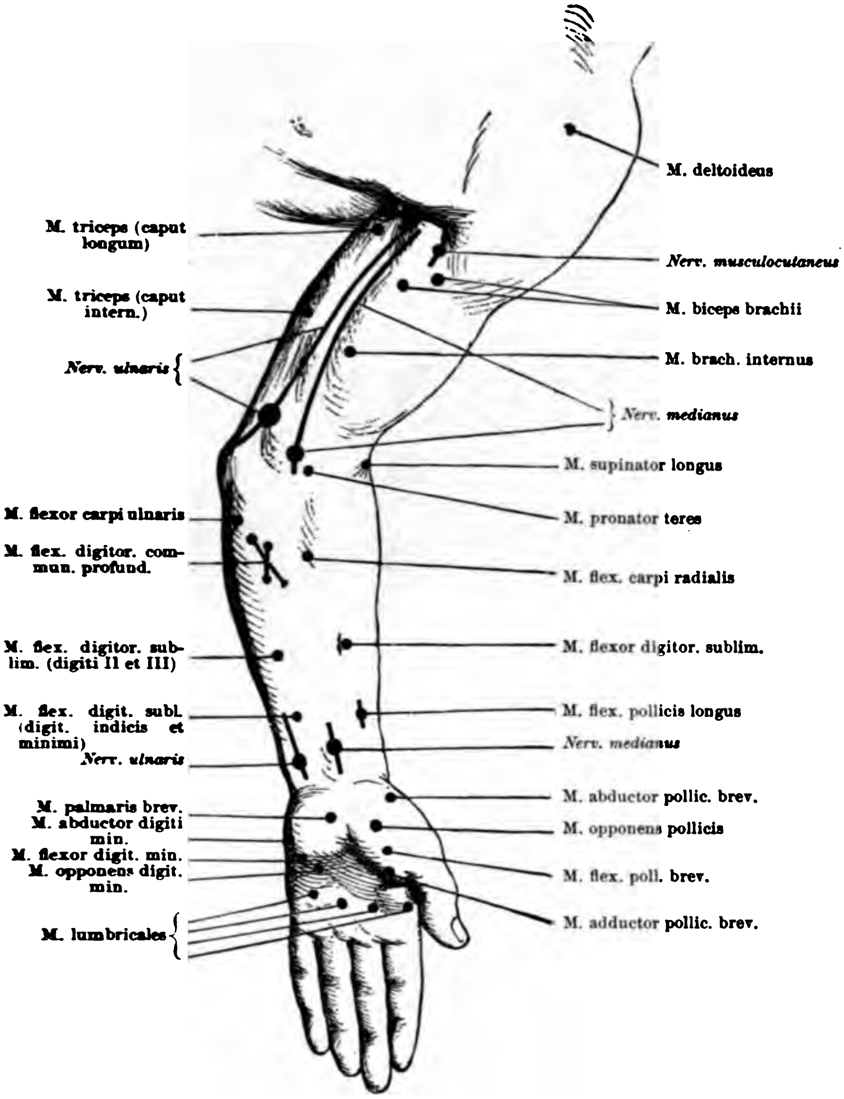

Fig. 9.—Nerves and motor points in upper extremity.

that to base the record upon the number used is futile. Where a rheostat is employed in controlling a lighting current of a given potential, an index scale can be arranged that will furnish fairly satisfactory records.

A uniform method of making electric tests is important. The arrangement of electrodes recommended by Erb and found satisfactory in all instances is perhaps the best. The large or *indifferent electrode* thoroughly moistened in warm water, with which it is also well to wet the skin, is applied over the sternum, where the absence of muscles and of much sensitiveness obviates disturbing features. The small or *active electrode*, fitted with an interrupter and well moistened, is then placed over the nerve or muscles to be examined, and the current allowed to pass (that is "*made*") or is interrupted (that is, "*broken*") as required.

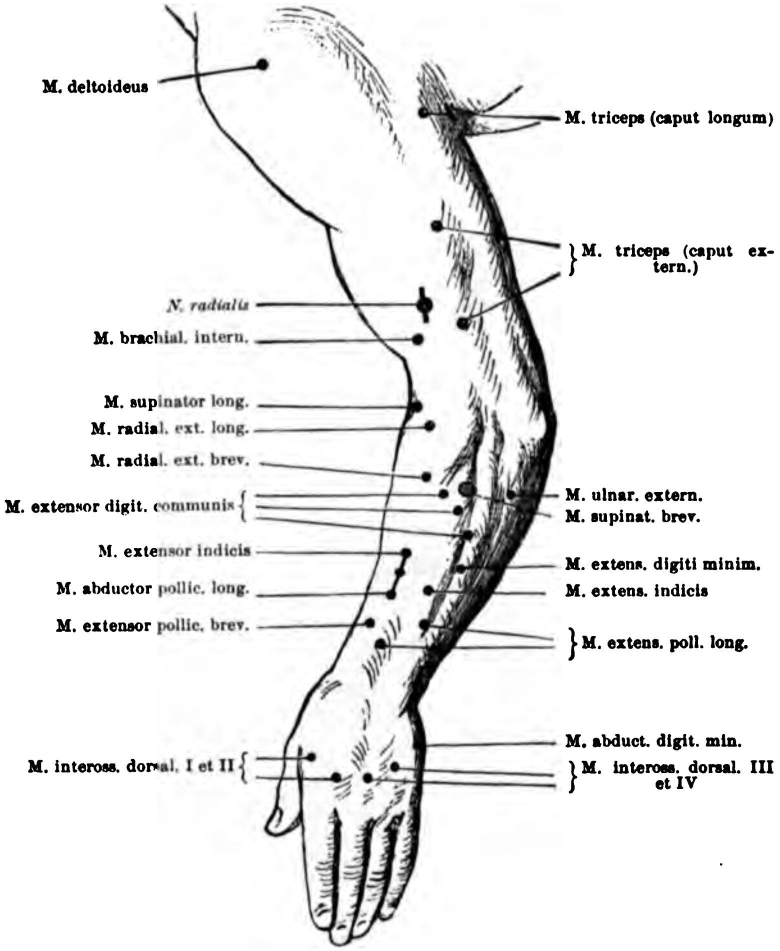

Fig. 10.—Nerves and motor points in upper extremity.

This interference, for the sake of brevity, is called the *make* and *break* of the constant current.

In Health.—The *nerve-trunk*—as, for instance, the ulnar at the elbow—in a healthy person, when stimulated by a mild *faradic* current, causes all the muscles to which it is distributed to contract more or less vigorously in proportion to the intensity of the current employed. It is to be particularly noted that upon allowing the current to pass the response is instantaneous, and that the muscular contraction at once reaches its maximum and maintains it until the current is modified. This phenomenon attends stimulation by either the positive or negative faradic pole or electrode, but the negative causes a slightly stronger effect.

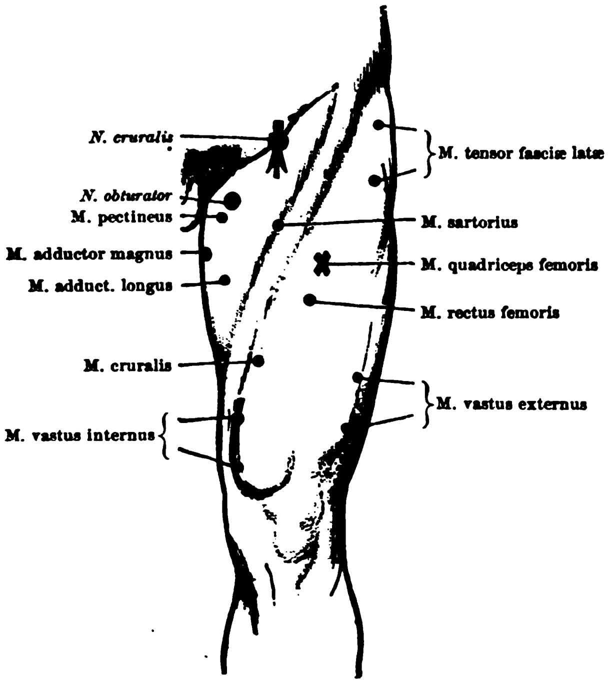

Fig. 11.—Nerves and motor points in lower extremity.

If, now, the *constant current* be substituted, we notice, first of all, that mild currents applied to the *nerve-trunk* produce no response. When the current is sufficiently increased it causes, only at the make and break, single instantaneous contractions of the innervated muscles, which as quickly subside. Looking closer, we notice that the contraction with the negative make—otherwise the cathodal closing contraction (C. C. C.)—is the first to appear as the current is gradually increased, that next comes the positive make, or anodal closing contraction (A. C. C.), followed in turn by the positive break, or anodal opening contraction (A. O. C.), and then, finally, by the negative break, or cathodal opening contraction (C. O. C.). Further, that by the time we have C. O. C., A. O. C.

will be vigorous, A. C. C. very strong, and C. C. C. produces a constant tetanic condition lasting as long as the current flows when once it is set up by the make,—a condition called cathodal closing tetanus (C. C. T.).

The contractions from stimulating the nerve-trunk thus in health appear in the following order: C. C. C., A. C. C., A. O. C., and C. O. C., and this indicates their intensity in a decreasing order. The important point to constantly bear in mind is that the negative make contraction through healthy nerves is stronger than the positive. This fact is thus formulated: C. C. C.>A. C. C.

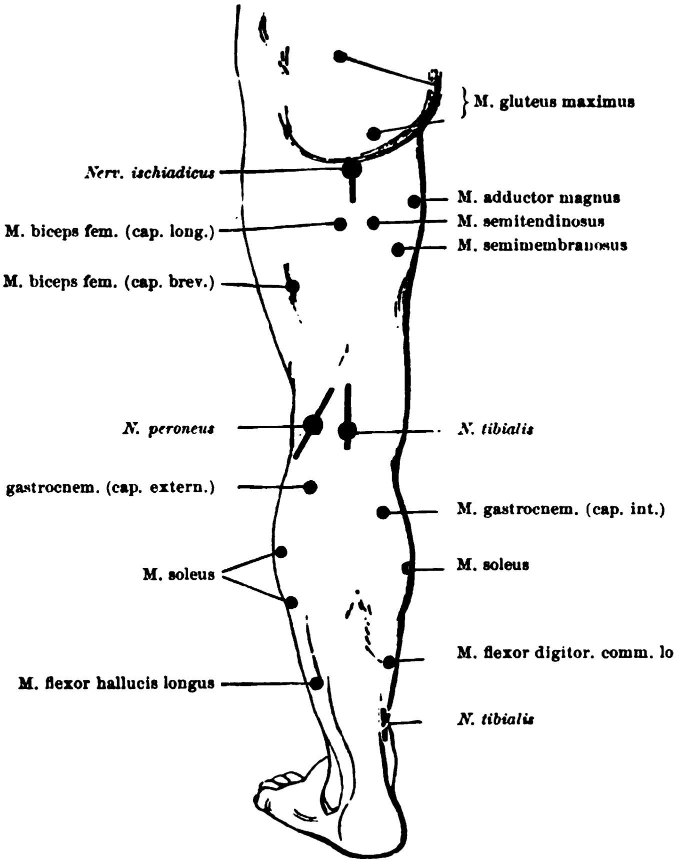
M. gluteus maximus
Nerv. ischiadicus
M. adductor magnus
M. biceps fem. (cap. long.)
M. semitendinosus
M. semimembranosus
M. biceps fem. (cap. brev.)
N. peroneus
N. tibialis
M. gastrocnem. (cap. extern.)
M. gastrocnem. (cap. int.)
M. soleus
M. soleus
M. flexor digitor. comm. longus
M. flexor hallucis longus
N. tibialis

Fig. 12.—Nerves and motor points in lower extremity.

When the active electrode is applied over the belly of a given *muscle*, a muscular contraction is set up by the *faradic* current, the same as that produced by stimulating the nerve-trunk. If the current be not too strong and the electrode not too large, the effect may be limited to the given muscle, or to a portion of a muscle. Every muscle may also be actuated by the *galvanic* current by applying the active pole on the point corresponding anatomically to the entrance of its motor nerve. A familiarity with *motor points* is of practical importance. They are shown in the accompanying diagrams. Slight variations in their location are common, and several trials are often required to find them. It is well to mark with a lead-pencil the necessary points, if repeated tests are to be employed. The galvanic *polar responses* are, in healthy muscles, identical with those obtained through the nerve-trunks.

In Disease.—Diseases changing the nutrition and structure of the lower neuron, which consists of the spinal cell-body and its peripheral projection and termination in muscular tissue, alter not only the trophic conditions over which the neuron presides, but also interfere with its electric properties. If the spinal cell be destroyed, or its peripheral filament divided, degeneration follows below the lesion, in both the nerve and muscle, with loss of voluntary control. The reactions of such a nerve and muscle to electricity are modified in a highly characteristic manner. They present the *reaction of degeneration* (R. D.), the presence of which is an indubitable sign of structural change. At the end of about a week, during which there has been a gradual decrease of electric excitability in the muscle and nerve to both faradic and galvanic shocks, four differences become manifest:

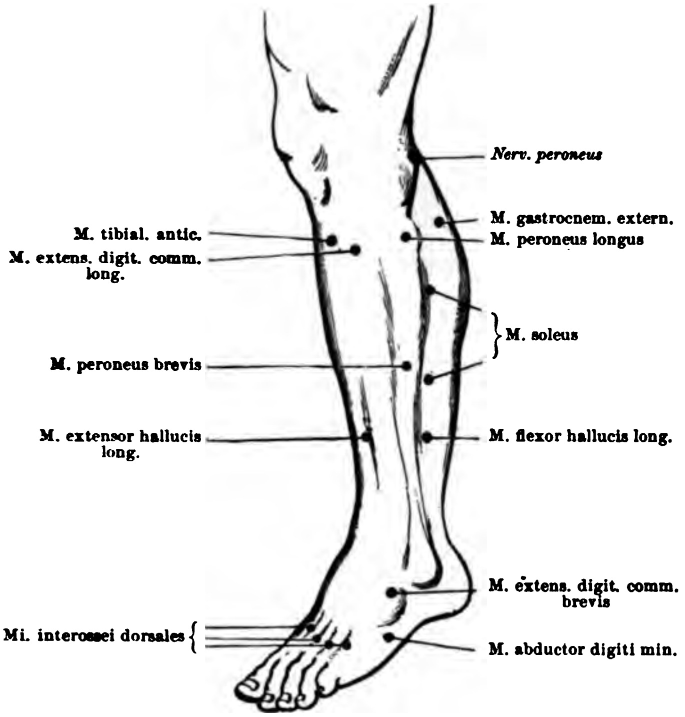

Fig. 13.—Nerves and motor points in lower extremity.

First. The *muscle* responds weakly, sluggishly, and deliberately to faradism, and shows a tendency to maintain the contraction after the current is withdrawn. This is the *modal change*.

Second. The *nerve-trunk* loses progressively and equally its responsiveness to both galvanism and faradism—a *quantitative change*.

Third. The *muscle* becomes much more excitable by galvanism and much less excitable by faradism, which latter reaction with the nerve-trunk responses is completely lost after two or three weeks. This is the *qualitative change*.

Fourth. A *polar change* appears in the *muscle* about the second week when directly stimulated by galvanism. It is now found not only that the muscle is more readily caused to contract by the constant current, but that the normal mastering strength of the negative closing contraction over the positive has disappeared and that the positive closing contraction is equal to or greater than the negative. Expressed thus: A. C. C. = or > C. C. C. Anodal tetanus (A. T.) is often obtained. This polar difference continues until the nerve either regenerates and regains its normal status, or, failing to recover, gradually the galvanic irritability subsides. It is entirely abolished with all other electrical responses in *complete degeneration*.

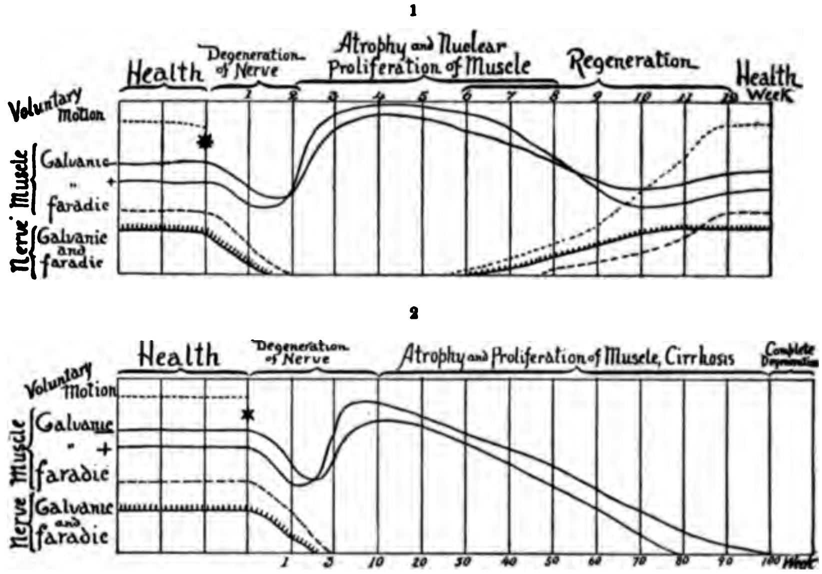

Fig. 14.—1, Paralysis with early return of motion (modified from Erb). 2, Incurable paralysis with complete atrophy and degeneration (modified from Erb).

Should recovery take place, the restitution is marked ordinarily, first, by a return of voluntary control, then, by the appearance of galvanic and faradic excitability in the nerve, and, last of all, by faradic excitability of muscle. The hyperexcitability of the muscle to galvanism and the polar variations from the normal gradually right themselves at the same time. These changes and the pathological process in nerve and muscle are shown in the diagrams (Fig. 14) modified from Erb.

In some cases we find a *partial reaction of degeneration*, the nerve-trunk response being only slightly modified. The inverted polar formula is not necessarily present. The *most essential element* in the reaction of degeneration is the modal change,—the slowly appearing muscular response,—so at variance with the instantaneous effect produced in health. Next in importance is the lessened faradic control of the muscle, and, last, the polar modifications. The variations in the galvanic and faradic responses in disease are due principally to the fact that currents of a certain strength and duration are required to actuate the changed sarcode cells, and the rapidly interrupted faradic current thereby becomes inoperative. The galvanic current, if interrupted with sufficient rapidity, is equally powerless.

In some conditions is found a simple *increase or decrease of electrical excitability*, constituting a pure quantitative change. In those diseases where the muscles seem permanently affected, as in the progressive atrophic myopathies, the responses are reduced, but are proportionate to the amount of healthy muscular fibers remaining. Some spasmodic conditions, like facial tic, present an increased activity to electric stimulation. There is increased electrical excitability in tetany and in Graves' disease. In myotonia we encounter a peculiar electrical response called the *myotonic reaction*. In this condition there is normal faradic excitability of nerves, but the electrical excitability of the muscles is greatly enhanced to all forms of current. The positive and negative closing galvanic contractions are nearly equal and all responses are tonic and prolonged. The mechanical excitability of the muscles, as to blows, is similarly exaggerated. In myasthenia gravis the muscular responses are rapidly exhausted by repeated stimulation with faradism or galvanism. They gradually lose force as the stimulus is repeated, and in a few moments fail completely, but reappear after a short rest. This peculiarity is called the *myasthenic reaction*.

The use of electricity to test cutaneous sensation is open to so many errors and attended by so much difficulty that simpler means, always at hand, are to be preferred. In those feigning complete cutaneous anesthesia the surprise of a strong, painful, faradic current from a dry, metal electrode will be likely to unmask the deceit.

When the active electrode is placed over the closed *eye* or at the temple, the make or break of constant currents is attended by a subjective sensation of light, and this test serves to show the activity of the retina and optic nerve. By placing the indifferent electrode on the same side of the neck, one eye at a time may be examined, and for this purpose only small currents—from two or three cells or of as many milliamperes—should be used. In the same way either *auditory nerve* may be tested by placing the active electrode over the mastoid or before the tragus, the negative pole producing much the louder sound.

For testing the nerves of *taste* the galvanic current is a prompt and reliable agent. By those of keen perceptions a difference between the positive and negative pole can be distinguished. For the ordinary purpose of taste testing, electrodes ending in probe-pointed metal terminals, or even the metal ends of conducting cords held to the tongue or pharynx, at once give rise to an acid or salty taste, which is quickly appreciated by the patient. One or two cells are all that is required, and one side of the tongue can be easily compared with the other in lateral lesions, or the tongue of a healthy individual will furnish a standard when needed.

For the purpose of localizing the various muscular areas in the sensori-motor zone of the cerebral cortex, when it is exposed surgically or otherwise, a mild faradic current is used. It is applied to the brain surface by two probe poles, preferably mounted on a single handle, and so arranged that their extremities may be fixed at any distance apart. Such a current thus applied causes, in the related muscular periphery, similar responses to those produced by faradizing the nerve-trunk, but the movement has more of a purposive or gesticulatory character. These responses also follow an extradural application of the electrodes.

CHAPTER VI.

SENSORY CONDITIONS.

Modifications of sensation are among the commonest conditions attending nerve-lesions and functional disturbance of the neural apparatus. It is well to have a prefatory understanding of the fact that sensation, of whatever variety or quality, is due to the appreciation of temporary motion and contact, or, in simpler terms, that all sensations are modifications of touch. It is the impact of luminous and sound-waves on properly qualified nerve expansions that gives rise to sensations of sight and hearing. The contact of odorous and sapid particles stimulates the sensations of smell and taste. The recognition of the various qualities of objects in contact with the skin gives us information of size, shape, temperature, hardness, smoothness, and a thousand physical properties of the external world.

Another general consideration is that any overstimulation of sensory nerves is painful. That which is so readily called the pain sense is but the subjective recognition of this overstimulation coupled by instinct and experience with the concept of harm. Thus the contact of a sharp point is at first so recognized and the sense of pain comes when the pressure is increased and the stimulation intensified. It can not be doubted, however, that pain and thermic sensations have courses in the spinal cord separate from tactile impressions. Extremes of heat, cold, pressure, noise, light, odors, and tastes give rise to pain or to discomfort analogous to pain. They cause automatic or conscious efforts of avoidance and self-preservation. It is also a matter of common experience that sensations, except of an extremely painful character, if long maintained, are more or less completely ignored. Therefore, to elicit sensation the motion or contact must be temporary or repeated after distinct intervals. Clothing to which we are habituated gives rise to little or no conscious sensation. With the long persistence of an odor there is a gradually diminishing perception of it. On the other hand, the sudden stopping of a clock or other familiar and disregarded sound is almost startling.

In considering the various qualities and modifications of the sense of touch it is requisite to bear in mind that keenness of sensibility varies greatly not only in different cutaneous areas in a given subject, but considerably in different individuals. The more keen, active, and intelligent the make-up of the subject, the quicker and more precise are the responses. To all tests of sensation the disadvantage pertains that we are dependent upon the responses of the person tested ; so that such tests are only partly objective. Sudden extreme pain, however, is usually attended by certain recognizable signs, such as change of countenance, quickened pulse, dilated pupil, pallor, and even perspiration. To carry out the needful tests we require the intelligent coöperation of the patient, and to this end he must understand exactly what is sought. In order that his replies may depend upon his receiving information through the tested source alone, the eyes should be bandaged or other suitable precautions taken to prevent their use. Check tests must also be used, such as asking " what is felt " when nothing is applied, or by using some indifferent object in place of the one which the patient is expecting, as the finger-tip, requiring him at the same time to tell what it is.

The attending conditions must be usual and natural. It is useless to test the sensibility of chilled extremities or to expect reliable replies from the stuporous. In many instances, moreover, there is much sensory disturbance without the patient being aware of it, as in hysteria; or there may be dissolution of the various qualities of the sense of touch that has not specially attracted his attention, as in syringomyelia.

The **tactile sense** enables us to recognize the contact of objects with the skin. In a crude way its delicacy may be tested by stroking with a feather or flake of cotton, by touching lightly the ends of hairs growing on the limbs, or by ruder contacts when the sense is found blunted. An instrument called an esthesiometer, consisting of two movable points, is frequently employed in testing this element of the sense of touch. A pair of compasses answers the purpose. Observations are made as to the least distances at which two blunted points are both recognized when brought into contact with the skin at the same time, and in a line parallel to the course of the nerve supplied to the part. These distances vary widely, as between the tip of the tongue or finger and the dorsum of the body. The following average measurements will serve as a standard of comparison, or if the sensory disturbance be limited to one side of the body, the opposite corresponding area will furnish a better guide. Differences, to be significant, must be about double those here indicated:

TABLE SHOWING ORDINARY DISTANCES AT WHICH TWO POINTS ARE RECOGNIZED.

Tip of tongue	1 mm. (1-25 in.)	Tip of toes, cheeks, eyelids	12 mm.
Tip of fingers	2 "	Temple	13 "
Lips	3 "	Back of hands	30 "
Dorsal surface of fingers	6 "	Neck	35 "
Tip of nose	8 "	Forearm, leg, back of foot	40 "
Forearm	9 "	Back	60–80 "
		Arm and thigh	80 "

Another means of testing the tactile sense is to place variously-shaped objects on the skin and to ask a description of them, or to trace letters and numerals with a blunt point. The so-called **stereognostic sense** is a complicated faculty, embracing many or all of the elements of tactile sense. All those perceptions of the qualities of an object received through the sense of touch make up a complex enabling its recognition if repeated with sufficient frequency. Thus we can with closed eyes recognize by touch all familiar objects. In certain brain-states, particularly those involving the sensori-motor cortex, and perhaps in a more limited sense the parietal area, the faculty is lost, constituting a state of *astereognosis* or, better, *stereoagnosis*.

The **pain sense** may be readily investigated by using a pricking point, but it must not be too fine. A pointed quill or toothpick answers well except in marked analgesia. The patient is required to state whether a touch or a painful prick is recognized. If the part examined be hairy, pulling on a few hairs with the fingers or a forceps elicits a sense of pain very effectively.

The **pressure sense,** which is not of great importance, is tested by placing on the skin objects of the same size and external character, such as balls of equal size but variously weighted. A spring instrument fitted with an index and a scale is also used. Where the extremities are thus tested it is necessary to so support them that no notion of the pressure be given by motions communicated to the joints and muscles.

The **thermic sense** can be quickly, though roughly, examined by first breathing and then blowing on a part. In order to estimate it more exactly it is best to use test-tubes filled with water at various known temperatures. Every test requires that considerable surface come in contact with the skin for a few moments, as the integument must gain or lose heat proportionately before the exact degree of temperature is appreciated. Within a few degrees of the ordinary body surface-temperature—namely, 80° to 86° F.—slight changes are not recognized in health. Below this to freezing and above it to about 150° F. a variation of two to five degrees is readily perceived. Inability to recognize temperatures from 60° to 70° as cool and 86° to 100° as warm may be considered abnormal. Such a condition is denominated *thermo-anesthesia,* and, when complete, *thermo-analgesia.* Those parts of the skin possessing the most acute tactile sense are also most sensitive to heat.

The term **muscular sense** is used in two ways:

First. It refers to the sensations arising in muscles themselves. In a painful degree this occurs in a cramp or in a tetanizing faradic contraction. The tension on muscles and tendons is no doubt subconsciously recognized by the coördinating mechanism, and it is only when these sensations are greatly intensified that we take conscious note of them.

Second. By *muscular sense* we refer to the ability to estimate the weight of various bodies. This ability is to so great an extent a result of practice and varies so widely among healthy persons that unless great differences of weight are not appreciated the test gives little information of value. Balls, such as those employed in the pressure test, can be used, or attempts made to repeat a fixed number on the dynamometer. When the sensation of tendons, joints, and muscles is blunted, not only is the muscular sense reduced, but knowledge of the position of the limbs is wholly or in part wanting and ataxia is present, as described under Errors of Motility.

In certain conditions, notably tabes, *sensation* is *retarded* and the reaction time is greatly prolonged. A painful impression, as by the prick of a pin on the foot, may not be recognized and responded to for several seconds. In such cases the transmission of all touch sensations is slow. We prick the patient, having instructed him to say "now" as soon as he feels the needle, and note the interval, which, in health, is a small fraction of a second only. The intelligence and promptness of the patient materially affect the apparent length of intervening time. In some instances a single prick is recognized as two or more, or a

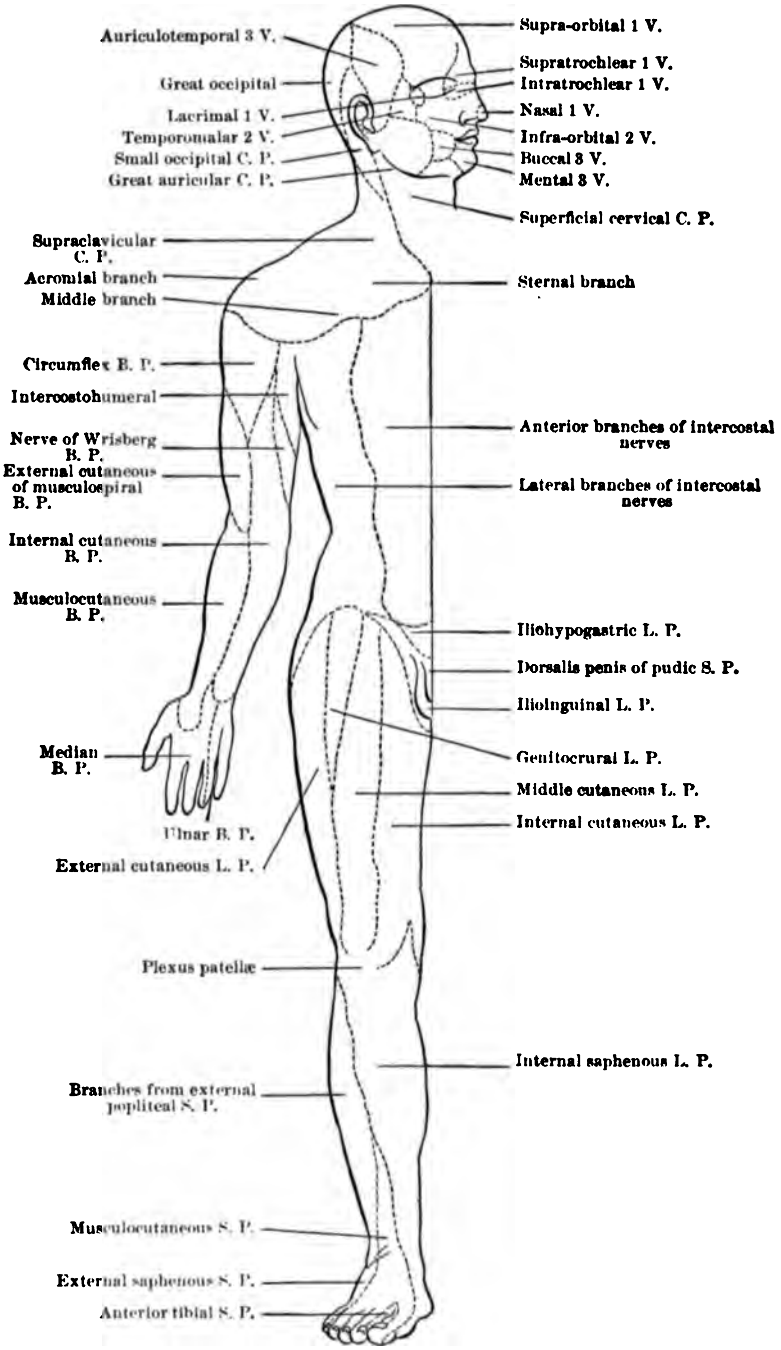

Fig. 15.—Cutaneous distribution of nerves (after Flower).

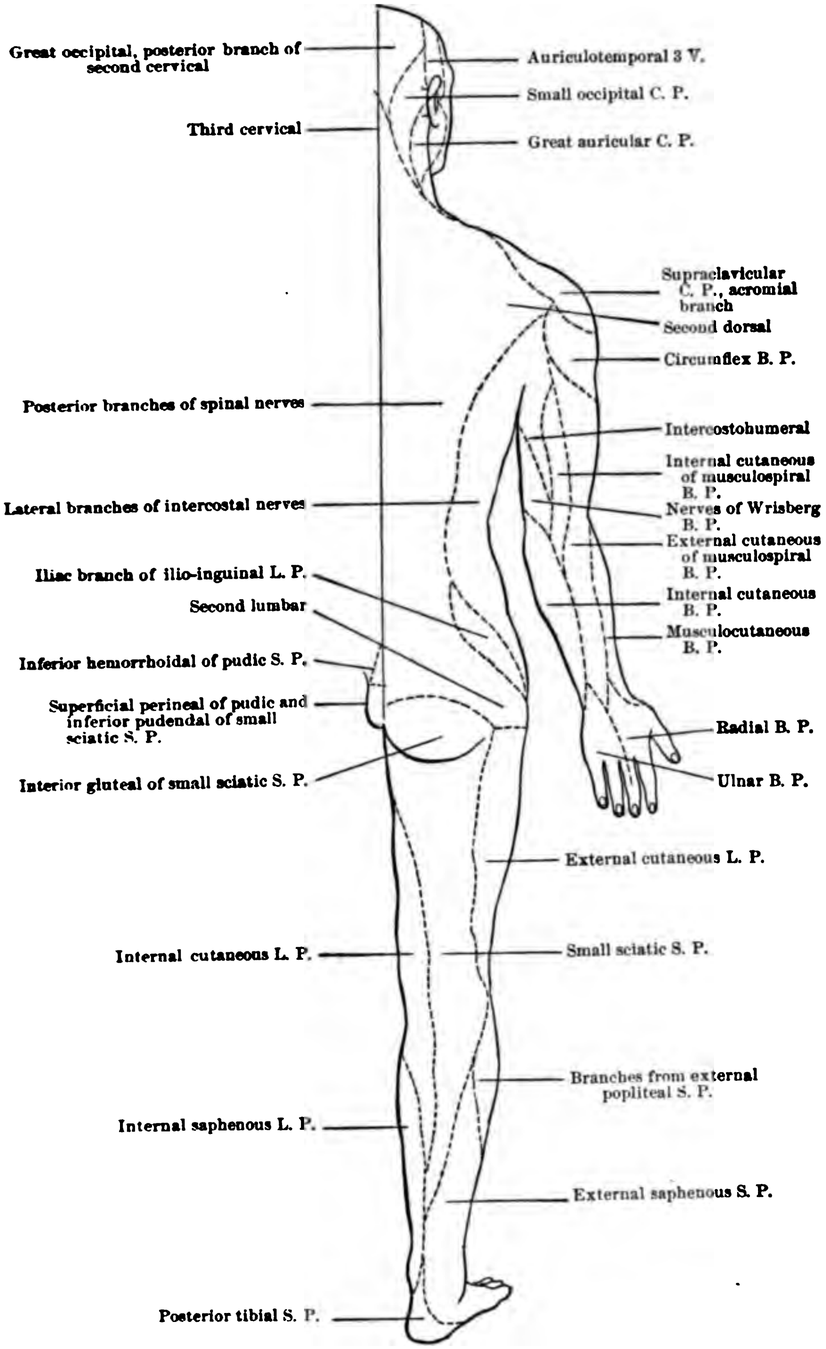
Great occipital, posterior branch of second cervical
Third cervical
Posterior branches of spinal nerves
Lateral branches of intercostal nerves
Iliac branch of ilio-inguinal L. P.
Second lumbar
Inferior hemorrhoidal of pudic S. P.
Superficial perineal of pudic and inferior pudendal of small sciatic S. P.
Inferior gluteal of small sciatic S. P.
Internal cutaneous L. P.
Internal saphenous L. P.
Posterior tibial S. P.
Auriculotemporal 3 V.
Small occipital C. P.
Great auricular C. P.
Supraclavicular C. P., acromial branch
Second dorsal
Circumflex B. P.
Intercostohumeral
Internal cutaneous of musculospiral B. P.
Nerves of Wrisberg B. P.
External cutaneous of musculospiral B. P.
Internal cutaneous B. P.
Musculocutaneous B. P.
Radial B. P.
Ulnar B. P.
External cutaneous L. P.
Small sciatic S. P.
Branches from external popliteal S. P.
External saphenous S. P.

Fig. 16.—Cutaneous distribution of nerves (after Flower).

painful impression is felt at a distant point,—a *referred sensation*,—or on the opposite side of the body at a symmetrical spot,—*allocheiria*.

The sensibility of *bones* and *joints* may be roughly but readily tested by resting the stem of a vibrating tuning-fork at those points where they are subcutaneous. Normally one perceives the communicated thrill.

Complete loss of sensation is properly termed *anesthesia*. Through usage this word signifies any degree of blunted sensation, and is qualified by adjectives such as partial, complete, or slight, as the case may require, and further limited by such combinations as muscle anesthesia, tactile, thermic, and joint anesthesia. The loss of the sense of pain is called *analgesia*, and this word is compounded in a similar way.

Sensation may also be intensified, giving rise to *hyperesthesia* and *hyperalgesia*. These conditions are made evident by the usual tests, and require no extended review.

In addition there are a host of purely *subjective sensory disturbances*, described as sensations of heat or cold, numbness, prickling, crawling, creeping, tingling, heaviness, deadness, etc.,—*paresthesiæ*. Areas so affected may show no alteration of sensibility when actually tested. Paresthesiæ usually are symptomatic of general nutritional states or of the so-called neuroses.

Having determined a localized *dysesthesia*, or condition of disturbed sensation, it is of the first diagnostic importance to outline it as accurately as possible. Sensation may be disturbed by lesions which involve sensory paths at any point from the cerebral cortex to the terminal organs in the muscles and skin. The dysesthesic area, however, presents a different and distinctive outline as various levels are injured.

If a *nerve-trunk* or branch be injured by traumatism or disease, anesthesia will be limited to the corresponding anatomical cutaneous distribution. *Per contra*, if such anatomical area be found to be anesthetic, the inference is at once justified that a peripheral lesion is present. As soon as the sensory nerve-fibers reach the spinal cord they ramify so widely in the various segments that it would be impossible for any spinal disease or injury to select a given number from all others. Figures 15 and 16 show these sensory areas, and should be carefully studied.

If the *spinal cord* undergo a complete cross-lesion or division, we have loss of sensation in the nerve-area below the upper level of the injury. Now, the distribution of anesthesia has relation to the cord-segments, and not to the nerve-trunks. For a practical understanding of this fact it is necessary to recall that the body, from one end of the vertebral chain to the other, is made up of a number of similar segments or links, all more or less perfectly represented in the skeleton, muscles, viscera, skin, and nervous apparatus. In the dorsal region the plan is comparatively simple, but as the limbs are reached it is greatly complicated. If vertical sections of the body were to be made approximately on these segmental lines, it would be requisite to place it in the all-fours position, making the coccygeal end of the vertebral column the posterior or last segment. The diagrams of Starr (Fig. 17) clearly show this, and the difference between the spinal-segment area and the nerve-trunk representation of sensation is apparent. It at once becomes plain, when we find an anesthetic area corresponding to a spinal-segment level that the lesion is in the cord and at a particular part of the cord; moreover, that its upper level corresponds to the upper level of the anesthetic zone. Just above this level, owing to the irritation of the sensory roots of the spinal nerves, there commonly is a band or girdle of hyperesthesia bordering the anesthetic area. This also serves to indicate the upper limit of the spinal lesion.

Should the cord injury involve only one lateral half of the transverse cord-section, the symptom complex of *Brown-Séquard paralysis* develops. The hemicordal lesion causes complete loss of power on the same side as the lesion in all parts below that point. There is also usually a slight loss of power below the lesion upon the opposite side. Anesthesia is complete on the side opposite the lesion, below its level. This is due to the fact that the sensory paths, upon entering the cord, cross to the opposite side, and then proceed upward. The sensory roots entering the cord at the point of lesion are destroyed; so that there is upon the same side of the body a band of cutaneous anesthesia which marks the exact level of the lesion of the cord. It varies vertically in relation to the extent of cord destroyed.

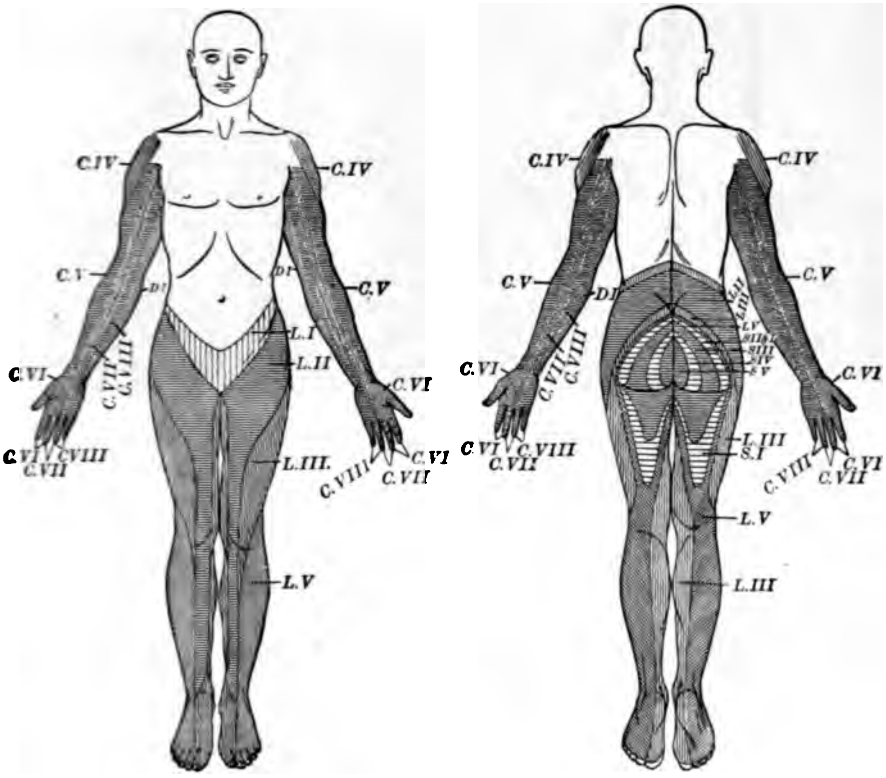

Fig. 17.—Cutaneous areas related to the spinal-cord segments (Starr).

The sensory roots entering the cord at the upper margin and at the lower limit of the lesion, irritated thereby, furnish, both above and below the band of anesthesia, a varying band of hyperesthesia on the paralyzed side. On the opposite or anesthetic side there is also a band of hyperesthesia due to the irritation of the sensory root as it crosses into the contralateral half of the cord at the upper level of the destructive process. This sensitive area is always a little below the hyperesthetic band of the paralyzed side. The accompanying scheme (Fig. 18) shows the sensory tracts and the involvement of the sensory roots upon each side. The distribution of sensory and motor disturbance is shown in figures 18 and 19. In addition, the reflexes are increased below the lesion on the paralyzed side, but abolished at the level and throughout the vertical extent of the lesion.

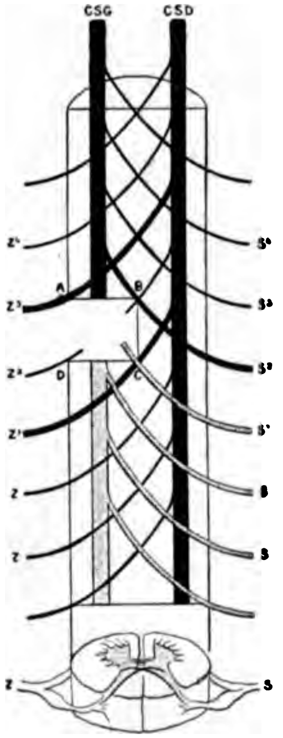

Fig. 18.—Scheme representing cord-lesion and effects in Brown-Séquard paralysis (after Brissaud). CSG, Left sensitive tract; CSD, right sensitive tract; A, B, C, D, lesion involving the left half of the cord; S, S, S, sensory roots from right side of body; Z, Z, Z, sensory roots from left side of body; Z^1, Z^3, and S^2 are irritated only at the points A, B, C, and their peripheral area is hyperesthetic: Z^2 is divided and its skin area is anesthetic on the same side as the lesion. Corresponding to S^1 and all the roots below arising from the right side of the body, there is anesthesia.

Lesions of the *internal capsule* involving its posterior sensory portion give rise to hemianesthesia embracing more or less accurately and completely one-half of the head and trunk with the limbs on the same side. It is usually associated with similarly distributed motor paralysis.

In the *cerebral cortex* cutaneous sensory representation corresponds probably quite closely to the motor fields. Cortical lesions in this field lead to paresthetic disturbances of sensation that have functional rather than anatomical limits, just as electrical stimulation of the cortex leads to purposive or grouped muscular movements, and not to those subserved by any spinal segment or nerve-trunk. In *hysterical anesthesia* a similar distribution is noted, the affected area often having the outline of the surface covered by a glove, sock, or sleeve, and limited with precision to the functional area that is the object of the patient's attention or solicitude.

Pain as a symptom is worthy of careful study. The first inquiry should be as to its *character.* Is it constant or intermittent? is it periodical? does it appear at a regular time daily, or every other day, or is it worse at any particular time of the day? Then, is it sharp, lancinating, or dull and heavy? Note exactly the *distribution* of the pain. Observe if it is limited to the cutaneous area of a peripheral nerve or if it has the distribution of a spinal segment.

In every instance of pain, seek for *tenderness*. The painful part, during the presence of protracted pain, is almost invariably sensitive. When lightly touched, certain portions of the hyperesthetic surface will be found especially affected. These *maximal points* of *pain* are usually situated where the sensory nerves become superficial or pass bony prominences.

It is best to carefully outline these sensitive areas with a blunt point, as the head of a large pin, and not trust to the rough gestures and statements of patients. In other instances the tenderness can only be detected by deep pressure. For instance, intercostal neuralgia produces superficial tenderness; pleurisy is marked by tenderness on deep pressure or percussion. To Dana and Head[1] we owe a definite knowledge of the relation of cutaneous sensitiveness and pain to visceral disease. Head's work, abundantly verified, clearly shows that the superficial surface of the body is in relation with the viscera through the spinal centers. Disease arising in the visceral portion of a vertebral segment gives rise to pain referred to the body or somatic area. Similarly, disease of the spinal segments gives rise to pain and other sensory disturbances in the skin, as already pointed out, and in the associated viscera. Mackenzie[2] insists that the viscera are insensitive and that pain is felt through the cerebrospinal nerves alone. That, for instance, the well-known tenderness over the liver readily elicited by pressure in disease of that organ is in fact confined to the skin and muscles of the abdominal wall. These areas, as mapped out by Head, with their maximal points for the body and limbs, are presented in figure 20 (*A*, *B*, *C*), and the following table shows the relation of the viscera to these areas:

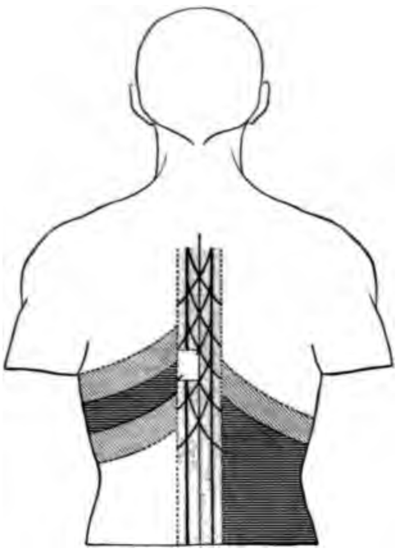

Fig. 19.—Application of the scheme to the trunk (Brissaud). Anesthesia is indicated by horizontal lines; hyperesthesia by diagonal lines.

TABLE SHOWING AREAS OF PAIN REFERRED FROM VISCERAL DISEASE.

Heart.—First, 2d, 3d, dorsal segments.
Lungs.—First, 2d, 3d, 4th, 5th dorsal.
Stomach.—Sixth, 7th, 8th, 9th dorsal; cardiac end from 6th and 7th.
  Pyloric end from 9th.
Intestines.—(A) Down to upper part of rectum.
  Ninth, 10th, 11th, and 12th dorsal.
  (B) Rectum.
  Second, 3d, and 4th sacral.
Liver and Gall-bladder.—Seventh, 8th, 9th, 10th dorsal.
  Perhaps 6th dorsal.

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[1] Head, "Brain," lvi.

[2] "Brain," 1902.

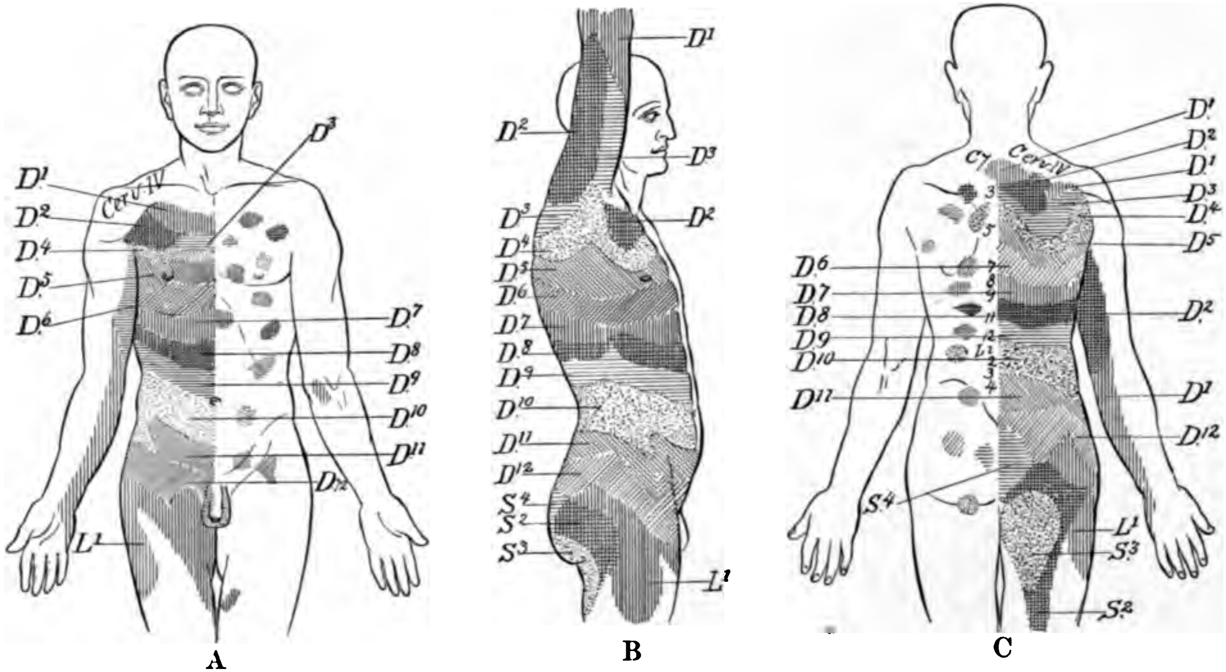

Fig. 20.—Cutaneous areas and maximal points of pain related to the vertebral segments (after Head).

Kidney and Ureter.—Tenth, 11th, and 12th. The nearer the lesion lies to the kidney, the more is the pain and tenderness associated with the 10th dorsal. The lower the lesion in the ureter, the more does the 1st lumbar tend to appear.

Bladder.—(A) Mucous membrane and neck of bladder.
First, 2d, 3d, 4th sacral.
(B) Overdistention and ineffectual contraction.
Eleventh and 12th dorsal and 1st lumbar.

Prostate.—Tenth, 11th, 12th dorsal.
First, 2d, 3d sacral and 5th lumbar.

Epididymis.—Eleventh and 12th dorsal and 1st lumbar.

Testis.—Tenth dorsal.

Ovary.—Tenth dorsal.

Appendages, etc.—Eleventh and 12th dorsal and 1st lumbar.

Uterus.—(A) In contraction.
Tenth, 11th, 12th dorsal and 1st lumbar.
(B) Os uteri.
First, 2d, 3d, 4th sacral, and 5th lumbar very rarely.

A further point elucidated by the same author is the relation of head pains and aches to visceral disturbances. Such pains and aches are marked by definite areas of sensitiveness when at their height and by ten-

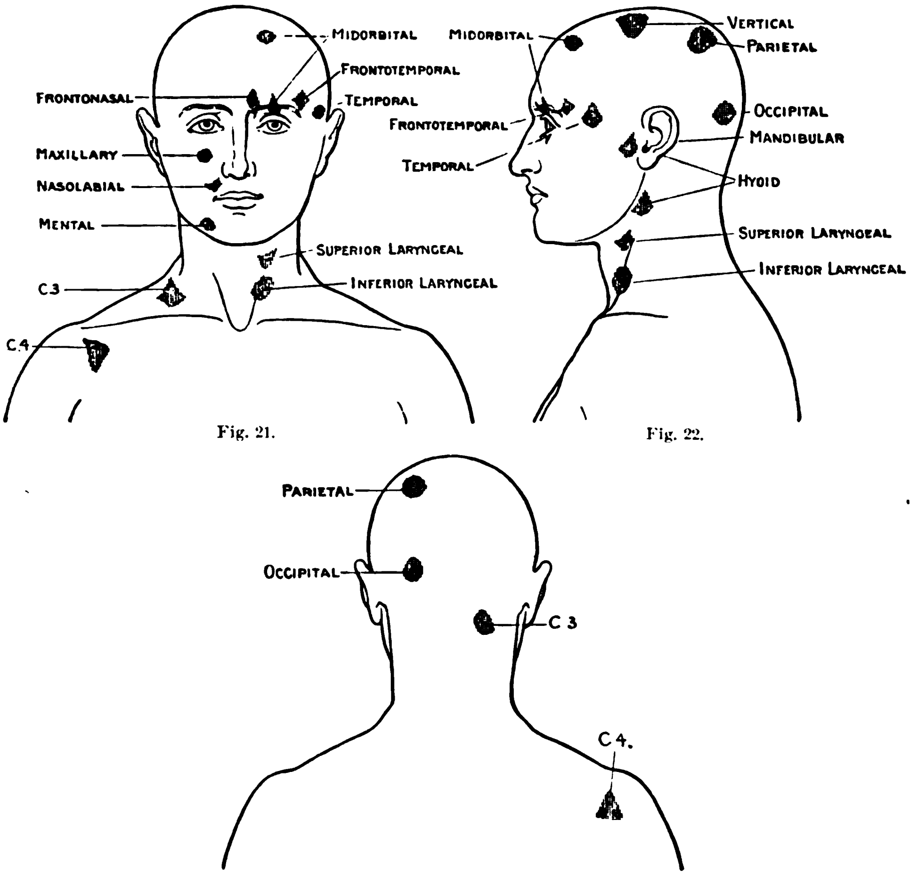

Fig. 21.

Fig. 22.

Fig. 23.

Figs. 21, 22, and 23.—Maximal points of referred and associated pain on the head and face.

der maxima. These maxima, as those upon the trunk, are practically the tender points of Valleix. They are much more persistent than the tender areas, which usually are only well marked when the pain is severe and gradually subside concentrically, leaving the maxima. On the head and face, as on the trunk and limbs, these pain-areas have segmental distribution and in no manner correspond to the peripheral nerve-supply.

Figures 21, 22, and 23 show the cephalic maximal points, and the following table shows the relation of the body-viscera to these head-points and also to the body-areas:

TABLE SHOWING ASSOCIATED PAINFUL AREAS ON THE HEAD RELATED TO VISCERAL DISEASE IN THE BODY.

AREA ON BODY.	ASSOCIATED AREA ON SCALP.	ORGANS IN PARTICULAR RELATION TO THESE AREAS.
Cervical, 3	Frontonasal (? rostral).	Apices of lungs. Stomach. Liver.
Cervical, 4	Frontonasal.	
Dorsal, 2.	Midorbital.	Lung. Heart. Ascending arch of aorta.
Dorsal, 3.	Midorbital.	Lung. Arch of aorta.
Dorsal, 4.	Doubtful.	Lung.
Dorsal, 5.	Frontotemporal.	Lung. Heart.
Dorsal, 6.	Frontotemporal.	Lower lobe of lungs. Heart.
Dorsal, 7.	Temporal.	Bases of lungs. Heart. Stomach.
Dorsal, 8.	Vertical.	Stomach. Liver. Upper part of small intestine.
Dorsal, 9.	Parietal.	Stomach. Upper part of small intestine.
Dorsal, 10	Occipital.	Liver. Intestine. Ovaries. Testes.
Dorsal, 11	Occipital.	Intestine. Fallopian tubes. Uterus. Bladder.
Dorsal, 12	Occipital.	Intestine. Uterus, etc.

In like manner disease within the head and neck has its referred pain, associated tender area, and maximal point. They may be thus tabulated:

ORGAN AT FAULT.	MAXIMA OF PAIN AND TENDERNESS.
Ciliary muscle (errors of accommodation) .	Midorbital.
Cornea	Frontonasal.
Iris	Fronto-temporal. Temporal and maxillary.
Vitreous (glaucoma) .	Temporal.
Retina	Vertical.
Teeth (upper incisors) .	Frontonasal.
Upper canine and first bicuspid . . .	Nasolabial.
Upper second bicuspid	Temporal or maxillary.
Upper first molar .	Maxillary.
Upper second and third molars .	Mandibular.
Lower incisors, canine, and first bicuspid .	Mental.
Lower second bicuspid	Mental or hyoid.
Lower first and second molars	Hyoid and pain in the ear.
Lower third molar . .	Superior laryngeal.
Membrani tympani .	Hyoid.
Middle ear	Vertical and behind ear.
Tongue, tip	Mental.
Tongue, lateral part .	In ear and hyoid.
Tongue, base	Superior laryngeal. Occipital.
Tonsil.	In ear and hyoid.
Nose, olfactory portion	Frontonasal and midorbital.
Respiratory portion and posterior nares .	Nasolabial.
Larynx	Superior and inferior laryngeal.

Brain disease presents pain of two varieties: First. When the meninges are involved there is a local pain with tenderness on pressure and percussion. Second. In conditions of intracranial pressure and disease of the brain proper there is superficial tenderness and the pain is widely distributed. In the second class of cases pain is usual in the brow, vertex, occiput, back of the neck and shoulder, and similarly distributed tenderness is common.

The *descriptions* of some pains by patients are classical and almost diagnostic. The lightning pains of tabes, the gnawing pains of rheumatism, the burning pains of neuritis, the girdle pains of spinal disease, the lead-cap pressure pain of neurasthenia, the pain under the breast and in the groin in hysteria, the daily recurring brow pain of malaria, and the nocturnal pains of syphilis have a significance quite their own. The *circumstances* under which pains occur often throw light upon their nature. Sciatica and lumbago are provoked by motion and allayed by rest. The head pains of eye-strain bear a distinct relation to ocular employment. Neurasthenic pains always increase under fatigue or depressing—that is, exhausting—emotions. A pain that is practically circumscribed has a tendency to spread to associated organs and to neighboring areas after long duration, general depression, or the onset of any marked physical illness. It thus becomes generalized.

CHAPTER VII.

THE SPECIAL SENSES.

Sight.—The eye presents many interesting and valuable symptoms in a wide variety of nervous affections. Its systematic examination should be a part of the case-taking in every instance.

The **lids** on the two sides may show a difference in the palpebral opening due to paralysis or spasm. Exact symmetry is the rare exception in health, but any marked acquired inequality, unless due to scars or local conditions, such as conjunctivitis, irritation, swelling, new growths, etc., implies some variation of muscular control or nerve-supply. A falling of the upper lid, or *ptosis*, is a common early symptom of tabes and syphilitic brain disease, while in a facial palsy an inability to close the lids is a marked sign. From the same cause the lower lid may be everted. An acquired prominence of the eyeball may greatly enlarge the palpebral opening. In exophthalmic goiter the lids frequently fail to follow the upward and downward excursion of the ocular globe. This is not entirely due to the protrusion of the eye, as it has been noted in the absence of this condition, and in some instances is congenital. In hysteria a condition often confounded with ptosis, but really an orbicular spasm, is sometimes encountered. *Blepharospasm* as a limited facial tic is a very common affection. Many states of brain and optic-nerve disturbance are marked by the closed lids of *photophobia*, which should not be confounded with that arising from inflammatory conditions of the lids, cornea, or iris.

Attention has already been directed to the reactions and reflexes of the *pupils*. In all examinations of the pupils the observer must exclude the actions of those drugs, like opium, cocain, and belladonna, which modify their size and reactions. Irregularities in their outline or inequalities in size are to be carefully noted, but pupillary deformities from antecedent inflammatory processes or injuries followed by synechia must not be mistaken for perverted innervation. Inflammation of the pleura,[1] apical tuberculosis, and pneumonia may cause either narrowing or widening of the corresponding pupil when tested by ordinary daylight. Paralysis of the pupillary muscles is *iridoplegia;* paralysis of the ciliary muscles—*cycloplegia*—is marked by the loss of the function of visual accommodation. They are usually found together, and then constitute *ophthalmoplegia interna.* Paralysis of the external muscles of the eye—namely, the recti, obliqui, patheticus, and the elevator of the lid—is denominated *ophthalmoplegia externa.*

The external muscles receiving their innervation from three sources, the third, fourth, and sixth cranial nerves, are very frequently involved singly or in groups. This gives rise to various deviations of the visual axes of the eyes or loss of power in directing them conjointly in some given direction, with resulting indistinctness of vision or complete double vision,—*diplopia.* Rarely a *monocular diplopia* is encountered as a pure hysterical symptom, but it may be the result of defective curvatures in the ocular media, as in corneal deformities. The special examination to determine the muscle or muscles at fault in these squint conditions will be taken up under the consideration of the diseases of the cranial nerves distributed to the ocular apparatus. Great and unwarranted stress has been put by some enthusiasts upon a condition of a lack of balance among the extrinsic muscles of the eye, named *heterophoria.* Of much greater importance are errors of refraction and accommodation in myopia, hyperopia, and astigmatism. They are attended by conscious or unconscious efforts at clear vision, constituting a condition of *eye-strain* that may furnish an active source of nerve waste. Extreme and constant deviations from the normal control of the extrinsic eye muscles can, no doubt, act in the same way, but slight variations in conditions of ill-health are commonly the result and not the cause of such states. As the general state fluctuates, they correspondingly vary for better or worse.

Vision can be readily tested by the types of Jaeger, and when seriously defective, by having the patient count fingers held in a good light against a dark background. Astigmatic error is roughly and quickly shown by the use of the numerous familiar charts for the purpose. For further details reference should be had to systematic works on the eye.

The *ophthalmoscope* is one of the most important instruments in the diagnostic outfit of the neurologist. An ability to readily examine the ocular fundus at the bedside or elsewhere is one of his most needful accomplishments. Familiarity with the normal ophthalmoscopic picture, supplemented by experience in recognizing *vascular disturbances, choked disc*, and *atrophy* of the optic nerve, will often render positive a host of otherwise obscure indications.

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[1] Chauffard, "Arch. Gén. de Méd.," Mar., 1905.

The *visual field* is the area over which objects are visible while the eye is fixed. In health its limits are tolerably uniform, being modified above and within by the brow and nose. In this field colors of objects are recognized by the normal eye at various distances and in a certain order from the *fixed point* upon which the gaze is directed. From without inward come white, blue, yellow, orange, red, green, and violet.

The form of the test-object is perceived before its color is apparent, as is shown by the chart (Fig. 24).

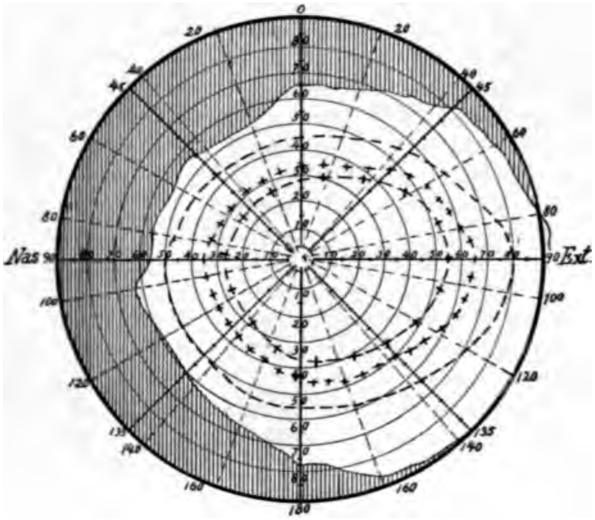

Fig. 24.—Normal visual fields for form and the various colors (after Souques). Form field unshaded. The blue field is inclosed thus — — — —, the red field thus + + + +, the green field thus — + — +.

The visual field is peculiarly modified by various diseases. In pronounced *hysteria* we have usually a concentric uniform reduction of the field. The color-fields may be reduced almost to the fixing-point or entirely obliterated. Even more characteristic is a rearrangement of them so that the red field overlaps or completely surrounds the blue. The relation of red and blue is therefore to be remembered. In *neurasthenia* the fields are frequently much reduced, and fatigue conditions promptly increase their contraction. In well-marked cases the efforts put forth by the patient in responding to the tests may serve to greatly increase the reduction of the fields within a few moments. In *tobacco* and *alcohol poisoning* and other toxic conditions the fields are sometimes greatly contracted and present blind areas, or *scotomata*. Destructive diseases occurring back of the globe may cut off a portion of the field, producing *hemiopia*, *scotomata*, *central blindness*, *concentric blindness*, or blindness in a *quadrant* of the field, as the fibers or centers related to the given area are involved.

To test the field of vision a perimeter is of service, and, for accurate examinations and records, indispensable. Roughly it can be done by placing the patient opposite a fixed point on a bare black or dark wall, at the distance of eight inches. With one eye covered he is ordered to maintain his gaze unswervingly on the fixed point. A small white object preferably about a centimeter in diameter, is brought into the field from the periphery along various lines radiating from the fixed point and the spot marked at which it is first perceived by the patient. By joining a series of such points the outline of the field is constructed and the various color limitations are similarly defined by noting the distances at which the color of the test-object is clearly recognized. Large blind spots may in this way sometimes be detected, the test-object being carried across the field to the fixing-point. The normal blind spot corresponding to the optic papilla must not be mistaken for a symptom of disease. More roughly still the field can be rapidly tested by facing the patient at a distance of about two feet. He is then directed to look you steadily in the eye opposite the one to be tested; that is, if the patient's left eye is under examination, he looks at the examiner's right. A small object is brought into the field of view in a plane midway between the patient and physician, and the distance at which it is recognized is noted. At the same time the examiner's own perception furnishes a check and measure to the test.

The subjects of *Daltonism*, or *color-blindness*, are congenitally defective in color perceptions. Some have complete *achromatopsia*, everything to them being probably of a neutral tint; others do not distinguish some elementary colors, as red from green; and others, again, fail to detect marked shades of the same color. This defect would modify tests of color-fields accordingly.

Hearing.—The sense of hearing is most often modified by local conditions in the meatus and middle ear. Our usual problem is to determine whether the nerve-apparatus is impaired. To this end we note at what distance on either side the patient can hear a watch, remembering that in advanced years such high-pitched sounds are not heard so readily as lower tones, like those of the voice. If the watch is not heard through the air, the ear should be closed by pressing in the tragus with the finger and the watch brought into contact with the root of the zygoma, the mastoid process, the parietal bone, or the teeth. If it is now heard, the difficulty is presumably in the conduction apparatus, and not in the nerve. This can be confirmed by using a tuning-fork in Rinne's test. Set it in vibration and place the handle against the mastoid or zygoma, the meatus being closed. When no longer heard, unstop the ear and hold the still vibrating fork close to it. The normal ear will detect tones through the air that do not reach it by bone-conduction, but if there be obstruction in the external or middle ear, the bone-path will be the more acute. The formula is B. C. > A. C. or B. C.=A. C. If there is no recognition of high or low tones by bone-conduction, the nerve-apparatus is undoubtedly diseased, or if with hearing greatly reduced A. C. > B. C. is still found, the probability is that the nerve is diseased.

Auditory hyperæsthesia is occasionally encountered in acute cerebral meningeal conditions and in hysteria. Severe headaches, meningitis, and many cerebral affections are marked by *dysacousia*,—sounds producing discomfort,—which may or may not be attended by real auditory hyperesthesia. In the relaxation of the tensor tympani muscle attending facial palsy low notes are heard with unusual distinctness, while those of high pitch may not be so clearly perceived as in health.

Subjective sounds, varying from an insignificant tinnitus to pronounced and formulated auditory hallucinations or loud explosions in the head, are referred to with great frequency by nervous invalids. Their starting-point is not rarely in the external or middle ear. Irritation of the auditory centers and nerve, however, is sometimes the cause.

Involvement of the *labyrinth* or of its nerves is marked usually by *vertigo*, and in extreme cases by *forced movements* in a given direction, which, as in Ménière's disease, may furnish attacks of great suddenness, throwing the patient to the ground. The *space sense* with equilibration is disturbed.

Finally, in no case of obscure brain symptoms should the possibility of extension of infection from suppuration in the middle ear and mastoid be forgotten,—a condition that specular examination and local searching should reveal.

Smell.—The sense of smell may be reduced or obliterated in one or both nostrils. It is necessary to test them separately, closing firmly the opposite anterior naris. The inspiratory efforts should not be too vigorous, as thereby the test odor may reach both nasal spaces through the pharynx. In selecting the test-material, pungent odors or irritants, such as ammonia or tobacco-snuff, should not be employed. In hysteria anesthesia of the mucosa may be associated with absence of the sense of smell, so that the strongest irritation gives rise to no response; otherwise, when the olfactory nerve is completely destroyed, stimulants and irritants have their usual effect. It is also well to choose an odor with which the patient is familiar, and to recollect that people vary greatly in keenness of scent. The sense of smell is greatly impaired by nasal catarrhal trouble, and is often practically lost during a severe cold in the head. Degeneration of the fifth nerve, which supplies common sensation to the nasal mucous membrane, also lessens its acuteness.

Occasionally the sense of smell is greatly intensified. Hallucinations of smell are rare, and in several cases have been found to depend upon disease in the temporosphenoidal lobe.

Taste.—Pure taste sensations are the recognition of bitter, sweet, sour, and salt. When a taste is associated with an odor, we speak of a *flavor*, and it requires the participation of the olfactory nerve, usually stimulated by way of the posterior nares. Flavors, in consequence, are lost with the loss of smell, and not with the loss of taste. The margin and tip of the tongue are more sensitive to sours and salts, while the base and pharyngeal pillars best recognize bitter and sweet. The entire gustatory area, which includes the dorsum of the epiglottis and even a portion of the rima glottidis, as well as much of the pharyngeal wall, distinguishes all tastes more or less readily.

For the purpose of testing taste, solutions of sugar, quinin, citric acid, and salt, or the powdered substances, answer. The tongue should be protruded and the test-substance applied to a small area. Some moments are usually required before the taste is perceived. The galvanic electric current furnishes a simple and reliable agent. Two probe-pointed metallic electrodes a few millimeters apart are placed on the portion of the tongue to be tested, and a non-painful current from one or two cells, of a few milliamperes' volume, is used. A metallic taste is elicited.

Taste is lost from the tip of the tongue in lesions of the facial nerve involving the chorda tympani, and from the rest of the gustatory area in lesions of the fifth nerve involving its lower division close to Gasser's ganglion. Hemiplegic states rarely show a one-sided loss of taste, while the hemianesthesia of hysteria, when pronounced, is usually marked by *ageusia* on the same side.

Perversions of taste—*parageusia*—and increased sensitiveness—*hypergeusia*—are sometimes encountered in neurotics and hysterics. Subjective taste sensations are also rare, but may furnish the aura of an epileptic attack or be dependent upon local irritation of the trunks of the nerves of taste, as in ear disease affecting the chorda and facial nerve.

CHAPTER VIII.

SPEECH.

There are as many kinds of speech as there are avenues to consciousness and routes therefrom. We have spoken language, written and printed speech, gestures and emotional attitudes that portray ideas and serve as media for the communication of ideas. Consequently, speech may be modified by disease in innumerable ways as the successive levels of the nervous system are invaded, and every variety of speech may be disturbed either in its perceptive or emissive channel.

The emission of vocal speech requires mechanically the *coördinate action* of the mouth parts, the larynx, and the chest-muscles of respiration. *Malformations* of the nose, throat, mouth, and larynx are attended with difficulty in shaping the resonant chamber for the precise modifica-

Physiological Table of Consonants.

	Voiceless Oral Consonants.	Voiced Oral Consonants.	Voiced Nasal Resonants.
Labials	P	B W	M
Labiodentals	F	V	
Linguodentals	Th S	Th Z	
Anterior linguopalatals	Sh T	Zh D L R	N
Posterior linguopalatals	K H or Ch	G Y	Ng

tions of vocal sounds in the production of spoken language. Cleft-palate, closed nasal spaces, and tied-tongue are not uncommon conditions of this variety. They cause difficulty mainly in the pronunciation of the consonant sounds. If we are familiar with the physiology and mechanism of consonant production, we have the key to diagnosis.

For this purpose the preceding chart, slightly modified from Wyllie,[1] is of great importance. He divides consonants into voiceless and vocal, as their pronunciation is or is not devoid of laryngeal sound. By having the patient pronounce these letters, or phrases containing them, the seat of the difficulty is at once recognized. For instance, in the palatal palsy of diphtheria the posterior linguopalatals and the nasal resonants, especially Ng, which depend on the separation of the nasal spaces from the pharynx by the soft palate, are slurred or lost.

Stammering, or **stuttering,** is a speech defect attended by difficulty in attacking properly the enunciation of words beginning with certain consonants. These consonants are reiterated with more or less spasmodic respiratory effort, and finally the word either drops out or is explosively spoken. There is always more or less incoördination in the needed muscular combinations.

Speech in idiots and infants often consists of the iteration of a syllable or single sound (*lalling*) or the repetition of the final word or phrase overheard by them, without reference to its meaning,—*echolalia*. In multiple sclerosis the speech becomes deliberate and each syllable is pronounced with the distinctness of *scanning*. In general paresis the words are jumbled. The patient catches in the middle of words, repeats syllables, slurs sounds, and omits terminals or even important words. This is called *syllable stumbling*. In *hereditary ataxia* the speech shows the incoördinate control of the musculature of vocalization and is usually slow, monotonous, and unmodulated. In cerebral palsies with athetosis speech may be characteristically modified by the spasmodic actions of the muscles of the tongue, throat, and chest. It is explosive, sputtering, now slow, now fast, and the tone qualities are uncontrolled. In *hysteria* persistent *aphonia* is a frequent symptom, a whispering voice only remaining. Complete *mutism* may also develop slowly or suddenly in this malady, but voice sounds, as in coughing or sneezing, usually remain to show the neurotic character of the disturbance. In *progressive bulbar palsy* the paralysis of the tongue is early marked by indistinctness of speech and a loss of the lingual consonant sounds. If the lips are weakened, the labials can not be produced, and, finally, through paralysis of the tongue and larynx, vocal speech is reduced to inarticulate noises.

In diseases marked by tremor, as in *alcoholism*, *Graves' disease*, and *paralysis agitans*, the voice is also tremulous. Depression, excitement, and the emotions generally are manifest in the timbre and modulation of the voice. The deaf are inclined to speak in a monotonous, high, or more often low, tone that is quite peculiar to them.

Aphasia.—Organic disease of the brain, throwing out of operation the various cortical centers related to speech or breaking up their connecting channels, produces peculiarly interesting phenomena that require very careful examination. Any of the qualities or varieties of speech may be affected, or almost any combination of defects may be present in a given case. Practically we have to investigate both spoken and written speech and to determine first how they are received and apprehended, and, second, how conceived and expressed. In other words, we try to determine whether the difficulty lies in the entrance-channel or the receptive center on the one hand, or in the formulating center and the emissive route on the other.

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[1] "Disorders of Speech," Edinburgh, 1894.

Take, first, the *reception of spoken speech.* Is the hearing good, tested by watch, tuning-fork, voice, and various sounds? If so, does the patient understand the words used; or is he *word-deaf*—that is, while hearing words does he fail to appreciate their meaning? Test this by directing him to execute certain movements—to shut his eyes, clap his hands, etc.

Second, how does he *produce spoken speech?* Is it reduced to a single expletive or phrase, or is he completely dumb? Does he forget names of common objects (*amnesia verbalis*), stammer, slur, stumble, or reiterate? Does he miscall objects with which he is familiar (*paraphrasia*), and is he aware of his mistakes? Is his speech a jargon of meaningless sounds or words strung together like beads? If dumb, can he write his answers? and if he can not write, can he indicate with his fingers the number of syllables in the names of objects pointed out to him? Finally, can he repeat or echo what is said to him, or is he inclined to echo his own words or expressions? does he understand his repeated words?

Written Speech.—If vision is good, does the patient understand written or printed questions? This can best be determined by writing simple commands, as, stand up! sit down! give me your hand! and not by questions that can be answered by a nod. Proper responses show comprehension. If the written questions or commands are only partially understood, we must attempt, by repeated tests, to decipher the limitations of his *alexia.* Secondly, does he write? If *agraphia* is not present, does he use wrong nouns (*paragraphia*), repeat letters or words, or make serious omissions? Can he write from dictation and from copy, and does he then understand what he thus writes?

When other speech avenues of the mind are blocked or only partly obstructed, the recognition of *gestures*, their use and repetition, should be noted. Some patients do not recognize gestures (*amimia*), or employ wrong gestures in attempting to explain themselves (*paramimia*).

To some patients objects have lost their meaning, so that familiar things and intimate friends are not recognized,—a condition called *mind-blindness.* The sense of touch (*stereognosis*) may still furnish information to the mind that has lost its recognition of visual impressions, so that a piece of money or familiar object unrecognized by sight may be correctly named when placed in the hand, though this faculty is also commonly defective when there is mind-blindness.

The handwriting, especially with the pen, in many cases furnishes diagnostic evidence of great value, as well as a means of studying the progress of the disease. A hand-magnifier will often bring out peculiarities that are not readily seen by unaided vision, and for the same purpose photographic enlargement may be used. When abnormalities are slight or only suspected, a specimen from something written several months or years previously will serve as a proper basis for comparison. Appropriate allowances for youthful growth or the decrepitude of age must be made. The education, habitude of writing, and physical condition at the time are also to be considered. As a rule, in health the down strokes are made with more strength, precision, and rapidity than the other written lines. If they show inequalities, tremor, waves, or marked angularities, the significance is greater than the appearance of these anomalies in the upward lines or connecting curves. The signing of the patient's name, for those who write it frequently, becomes quite automatic, and often fails to fairly show the character of the disturbance. It is well to dictate some ordinary sentence, to have the patient copy a paragraph from a newspaper, or write a little account of his illness. Many times it is only after writing a few minutes that the difficulty manifests itself. This is particularly true in writers' cramp. In general paresis the first of a letter page may be well, firmly, and coherently written, the latter part showing tremor, inequalities, omitted words, and incoherence. Blots, spatters, and wavering lines demonstrate the ataxia of the patient. The aphasic shows his cerebral lacunæ by using wrong words, by writing jargon, and by the repetition of letters, syllables, and words or phrases when not intended.

Mirror-writing is a variety in which the letters are formed backward, like printers' type, and appear properly when viewed in a reflecting surface. Left-handed children often write thus naturally, and it has been noticed in hysterics and degenerates. Rare cases of *mirror-speech* have been recorded, in which words were inverted by syllables or literally.

Varieties of handwriting are given in the description of the various diseases which present such peculiarities.

Finally, *photography* furnishes a most valuable adjunct to case-taking when any peculiarity of conformation, attitude, gait, or facial expression is observed. Serial photographs vividly present the course of the disease. The use of a *case-book*, properly prepared, is of the utmost value to systematize the examination and secure a full but concise record. The form given on page 70 may serve as an outline, requiring to be properly spaced for actual use.

FORM FOR NEUROLOGICAL CASE-BOOK PAGE.

Name .... *Nationality* .... *Date* ....
Residence .... *Age* .... *Civil Condition* .... *Children* ....
Occupation .... *Heredity* ....
Health History ....

Injuries ....
Habits ....
Present Illness ....

PRESENT CONDITION.

Alimentary System
Circulatory System
Respiratory System
Tegumentary System
Genito-urinary System
Pulse ***Temperature*** ***Weight***
General Appearance
Attitude
Gait

Motor Condition

Sphincters

Sensory Conditions

- ***Subjective***
- ***Objective***
- ***Muscular***
- ***Delayed***
- ***Thermic***
- ***Diminished***
- ***Intensified***
- ***Pain***

Electric Conditions

Trophic Conditions

Diagnosis

Treatment

Eyes

- ***Lids***
- ***Pupils***
- ***Movements***
- ***Vision***
- ***Field***
- ***Fundus***

Ears

- ***Deformities***
- ***Hearing***

Sense of Smell

Mouth

- ***Lips***
- ***Tongue***
- ***Palate***
- ***Articulation***
- ***Voice***
- ***Aphasia***
- ***Deglutition***
- ***Taste***

Brain

- ***Memory***
- ***Logical Powers***
- ***Emotions***
- ***Consciousness***
- ***Vertigo***
- ***Sleep***

Spinal Cord

- ***Superior Reflexes***
- ***Deep Reflexes***
- ***Coördination***
- ***Spinal Column***

Handwriting

PART II.

DISEASES OF THE CEREBRAL MENINGES AND CRANIAL NERVES.

CHAPTER I.

THE CEREBRAL MENINGES—PACHYMENINGITIS AND PIAL HEMORRHAGE.

Anatomical Considerations.—The coverings of the brain are admirably suited to protect it from injury and infection. Guarded externally by the skull and the scalp-pad, it is intimately enveloped by the dense, fibrous dura mater in a practically sealed sac. The usual anatomical descriptions of the cerebral meninges are misleading. Ordinarily three distinct membranes are named and described, when in reality there are but two. Lining the skull we have the *dura mater*, serving as an internal periosteum for the cranial bones and furnishing in part their vascular supply. It is entirely free from the brain, but gives off sheaths to the cranial nerves and the large vessels at their exit from the skull, and supplies venous channels or sinuses for the return circulation of the encephalon. The dural fold between the cerebral hemispheres and the tentorium cerebelli afford support and protection.

In normal conditions the brain fills the cranial cavity fully, and its soft covering membrane is everywhere in contact with the inner surface of the dura. The interval which separates them is called the *subdural space*. No actual space, however, exists, the two membranes being smoothly applied to each other and only separated by disease or mechanical means.

Closely investing the brain is the *pia mater*, made up of two layers or membranes very loosely attached by delicate meshes of fibrous tissue. The outer can be easily stripped from the under layer, and constitutes what is usually described as the arachnoid. The alleged double layers and spaces of the so-called arachnoid can not be demonstrated and do not exist. This outer pial layer we may call the *arachnopia*. Between it and the under layer, or *visceral pia*, is a varying space, the *pial space*, filled with a delicate, open, reticular network of fibrous tissue containing cerebrospinal fluid or lymph. It is an enormous lymph-space. At the gyral grooves the visceral pia dips to the bottom of the sulci. It everywhere closely adheres to the brain-cortex, which it follows through the transverse fissure into the ventricular chambers, furnishing the velum interpositum and bearing the choroid plexuses. The *arachnopia* bridges over the sulci. This arrangement at the great fissures and at the base of the brain in the intervals between the cerebrum, cerebellum, and medulla forms lymph-reservoirs, which are continuous with the pial spaces of the spinal cord through the foramen magnum. Delicate processes of the pia also accompany the cranial nerves and vessels from the skull, and are continuous with the extracranial lymph-channels.

Between the pial layers the cerebral vessels ramify. As they penetrate the cortex both arteries and veins are accompanied by delicate sheaths of the visceral pia, which form the perivascular lymph-spaces of the brain. Some of the great pyramidal cells of the cortex are encapsuled by diverticula from these perivascular lymph-channels, and are thus nourished. In a manner, therefore, these most important cortical elements may be considered as appendages of the pia, with which they have such intimate anatomical relations. The pia also pushes up the Pacchionian bodies into the vascular area of the dura near the venous sinuses at the vertex. These bodies are supposed to furnish an outlet for the meningeal lymph.

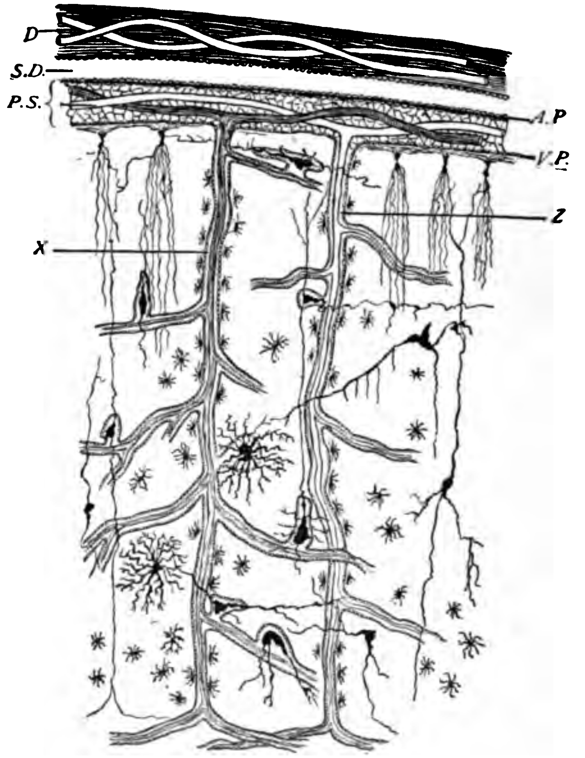

Fig. 25.—Diagram of cerebral meninges and cortex. *D*, dura mater; *S. D.*, subdural space; *P. S.*, pial space; *A. P.*, arachnopia; *V. P.*, visceral pia; *X*, arterial perivascular space; *Z*, venous perivascular space (Tuke).

The brain is constantly changing in bulk through variations in vascularity due to mental or physical activity. This would be impossible were it not for the ready displacement of the cerebral fluid. Losses of brain-substance by atrophy or otherwise are mechanically compensated in bulk by an increase in the pial fluid and the hydrostatic balance is preserved. The wide ramifications of the pial structure through the substance of the brain, its lymphatic prolongations, and its ventricular relations make plain many of the symptoms of meningitis and account for the serious sequels of the disease.

INFLAMMATION OF THE DURA MATER—PACHYMENINGITIS.

The dura mater, though a tough, resisting, fibrous structure, is subject to inflammatory invasion. As the outer or inner surface is affected, we speak of *pachymeningitis externa* and *pachymeningitis interna.*

Pachymeningitis externa is not a clinical unit. When the outer surface of the dura becomes inflamed, it is always a secondary condition, the result of the extension to it of infection from adjoining structures. Fractures of the cranium attended by sepsis, osteitis, necrosis, and new growths in the cranial bones may be its starting-point. Suppuration of the middle ear sometimes propagates inflammation to the dural covering of the temporal bone. A cranial gumma may incite it. Usually it is limited in extent. Very rarely considerable accumulations of purulent material between the dura and the skull may strip the membrane from the bone and occasion cerebral disturbance by localized pressure.

When inflamed the dura becomes thickened and strong adhesions to the inner surface of the cranial bones are formed. The danger consists in a resulting sinus-thrombosis or in septicemia. The treatment is that of the surgical condition of which the pachymeningitis is the sequel.

Pachymeningitis interna, *pachymeningitis hæmorrhagica,* or *hematoma of the dura mater,* is a chronic inflammation of the inner surface of the dura mater marked by one or more hemorrhagic membranous layers.

Etiology.—The arterial changes resulting from alcoholism are frequently the cause of this condition. In undoubted instances the congestion following alcoholic abuse has determined the vascular rupture that furnishes the laminated membranes. In dements, and especially in paretic dements, it is a common post-mortem finding. Infectious maladies, the exanthemata, erysipelas, and cachectic states, especially those marked by purpuric conditions, as scurvy, sometimes lead to it. It is more frequent among men than among women, and appears, as a rule, early or late in life.

Pathological Anatomy.—Internal pachymeningitis is essentially characterized by the thickening of the dura and the deposition internally of laminated new membranes of hemorrhagic origin.[1] Raswedenkow[2] insists that the first change is proliferation of the epithelial layer followed by fibrinous exudation and the formation of thin-walled capillaries and that the primary condition is a toxemia. Barratt[1] thinks that intravascular separation of fibrin is a constant initial feature followed by the other changes and finds such resulting membranes free from bacteria. These layers vary in number from two or three to as many as twenty, and in consistency from that of freshly extravasated blood to tough, well-organized, leathery membranes. They present, according to their age, the colors of blood under similar circumstances elsewhere. The new ones are bright red, the older ones yellow. They are only slightly adherent to the dura and to one another by fibrous connections, and are quite vascular when of some age. Their blood-vessels are delicate, poorly developed, and readily degenerate, thus furnishing new hemorrhages, which separate the older layers or form new ones on the cerebral surface. Adhesions to the arachnopia are practically wanting. The cerebral convolutions are flattened if the stratified new formation attains considerable proportions, and the cranial bones may also present pressure atrophy. In children the ossification of sutures and fontanels is retarded. This form of dural disease is usually found at the vertex, in the distribution of the middle meningeal arteries over the motor zone, but occasionally the basal fossæ are involved.

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[1] Meyer, "Path. Report, Ill. Eastern Hosp. for Insane," 1896.

[2] "Ziegler's Beitrag.," Bd. xxvii.

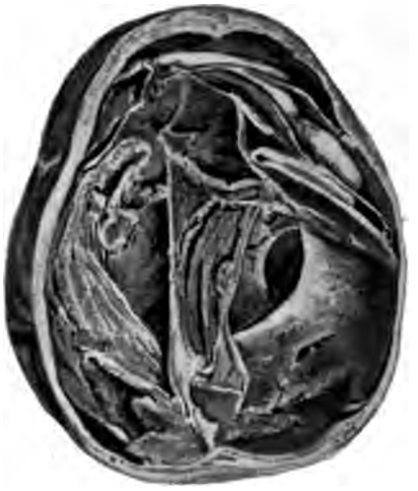

Fig. 26.—Pachymeningitis interna in a rachitic child with scurvy. The different layers, with cotton-wool pledgets interposed, are seen on the right side of the skull anteriorly (Southerland).

Symptoms.—The symptoms of the early stage before hemorrhages have occurred, and when the process is purely inflammatory, are very vague. In many cases no suspicion of the disease has been raised during life. The first recognizable symptoms attend the formation of a hematoma large enough to produce cerebral indications. These consist of pain in the head, intellectual troubles, loss of memory, awkwardness in muscular movements, insomnia, vertigo, rarely vomiting, limited or Jacksonian convulsions, apoplectiform attacks, rigidities and monoplegias presenting remissions. The temperature is fickle and uninstructive. During the convulsive attacks it attains a high degree, but in the intervals may be subnormal, normal, or slightly elevated.

Course.—As the early symptoms escape recognition, the duration of the disease is indeterminate. It usually runs a protracted course, and may in rare instances terminate in recovery, with resorption of most of the new tissue. As it is practically an expression of a serious or hopeless underlying condition, the ordinary end is death. This may follow an apoplectiform seizure or a comatose condition, but usually is preceded at intervals by a number of such attacks, each of which leaves a certain trace behind it in the form of added mental or motor disability.

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[1] "Brain," 1902.

Diagnosis.—The diagnosis is difficult. In drunkards, dements, and cachectic children the appearance of the cerebral symptoms mentioned should call the disease to mind. A history of preceding apoplectiform attacks, with practically complete remissions, would strengthen the suspicion. The thickened membranes form anatomically a subdural tumor, and at times present all the symptoms of a new growth in that locality. The nocturnal cephalalgia of syphilitic meningeal involvement and other luetic features will usually differentiate that disease. The distinction from tubercular meningitis in children rests upon the absence of constipation, abdominal retraction, severe headache, rigidity of the neck, and intense respiratory and circulatory troubles. In adults, cerebral apoplexy usually is of more rapid onset, but cerebral thrombotic softening often presents a parallel symptom group.

Treatment consists of measures to relieve the basic condition. Alcoholic addiction, infectious diseases, and the cachexiæ having received appropriate attention, the various cerebral indications are met as they arise. Quiet, the ice-cap, elevation of the head, antispasmodics, cathartics, sinapisms, hot foot-baths, and other means to decongest the cerebral circulation will be of service during the convulsive attacks. Ergot should not be advised, as the bleeding vessels, devoid of muscular layers, would be placed at a still further disadvantage by the increased arterial tension. Monro and Ballard, of Boston, have reported a number of cases in adult alcoholics successfully treated by trephining and the evacuation of the clots. When the diagnosis can be made, such procedure is urgently indicated.

PIAL HEMORRHAGE.

Pial hemorrhage, or *meningeal hemorrhage,* takes place either outside of the arachnopia, on the inner surface of the dura, or within the meshes of the pia, or in both locations at once. The *extrapial* variety of meningeal hemorrhage may be found at all ages, but is most common in *the new-born.* In about one-half of the cases still-born children present this accident, apparently due to protracted labor, sometimes to instrumental delivery, and even to precipitate birth. In one-third of the cases of asphyxiated new-born, Cruveilhier claims that subdural hemorrhage is the main difficulty. The clots are commonly found over the convexity, and are, in surviving cases, a pregnant source of idiocy and cerebral palsy.

Later in life pial hemorrhage is usually produced only by extreme violence to the skull, as in concussing injuries or fractures. The hemorrhage may come from a dural sinus or from the meningeal arteries. It practically requires a fracture with displacement or a penetrating wound to cause sinus bleeding. The location of the middle meningeal artery in a bony channel at the anterior inferior angle of the parietal bone, a frequent seat of fractures and direct violence, renders it particularly liable to injury. In many cases the hemorrhage from the meningeal vessels takes place at the contre coup point. Certain debilitating and infectious conditions predispose to and may rarely cause subdural hemorrhage; for instance, hemophilia, purpura, small-pox, scarlatina, typhus, typhoid, and acute rheumatism. It is found in chronic alcoholism, especially after a debauch. A ruptured meningeal aneurysm may flood the subdural space.

The onset is acute and marked by apoplectic seizure and rapidly developing unconsciousness. In traumatic cases the patient not seldom rallies more or less from the first bewilderment of the concussion, and after a variable interval of minutes, or even hours, sinks into unconsciousness. The cortical irritation is manifest in one-sided or more circumscribed and repeated convulsions, rigidities, and tremors. The pupils dilate unevenly, the coma becomes profound, the pressure indications intensified, and the patient usually dies in from twenty-four to seventy-two hours, unless relieved by operation.

Intrapial hemorrhage takes place within the pial spaces, or strips the pia from the cerebral cortex. It is the usual meningeal hemorrhage of adult life, and its common location is toward or in the basal region.

The extravasated blood may widely infiltrate the pial spaces or merely form diffuse ecchymotic discolorations. In large quantities it may force itself into the ventricles through the transverse fissure, and even travel down into the pial spaces of the cord. Usually it forms a thin covering over the surface of the convolutions, dipping into and filling the intervening sulci. Under the hemorrhage the surface of the cortex often appears softened and lacerated. Ordinarily the blood is of arterial origin; rarely it comes from veins and very exceptionally from a ruptured sinus. Except in traumatic cases, disease of the vessels, such as aneurysm, miliary aneurysm, sclerosis, atheroma, fatty degeneration, or acute infectious softening of their walls, furnishes the essential element of causation. It follows that periarteritis and endarteritis are the initial steps in the process that eventuates in hemorrhage. The development of these conditions is taken up more at length under the diseases of cerebral arteries in Part III, to which the reader is referred.

The onset of this form of meningeal hemorrhage is also acute and apoplectic, with a rapid downward tendency toward death. There may be partial temporary restoration to consciousness, but the patient presents indications of great shock and feebleness. A recurrence of apoplectic symptoms is usually quickly followed by death. Previous indications of localized disease, such as aneurysm of the basilar or other large vessel of the base, may have been present in the form of cranial-nerve palsies with the usual features of endocranial tumors. The apoplectic seizure is usually less sudden and violent than in cerebral hemorrhage of the ordinary capsular variety, and often rather gradually develops the full apoplectic state and profound coma. Hemiplegia and conjugate deviation of the head and eyes have been noted in rare cases, but their absence is the rule, and aids in differentiating this condition from cerebral hemorrhage. Epileptiform and tetanic features are occasionally present and imply cortical irritation. The pulse may be slow and respiratory difficulty present. At first the rectal temperature is reduced, but returns to the normal and rapidly mounts as death approaches. This takes place, as a rule, in from a few hours to a few days. Rare cases have endured a week, and still rarer ones survived.

The diagnosis of intrapial hemorrhage is difficult. It closely resembles cerebral hemorrhage, cerebral thrombosis, and the hemorrhage arising from pachymeningitis interna. The chief symptoms on which reliance is to be placed are the rapidity of onset and the quickly increasing symptoms without paralysis and convulsions. An internal pachymeningitis usually presents a significant history of headaches, and occurs in a limited class of patients with gross degenerations. The differential features of cerebral hemorrhage and softening are tabulated in Part III.

The outlook is extremely grave and death is almost certain to terminate the case. In these days of aseptic cranial surgery an exploratory opening is allowable, and furnishes practically the sole means of controlling the hemorrhage and saving the patient.

CHAPTER II.

INFLAMMATION OF THE PIA MATER.

Leptomeningitis, *acute cerebral meningitis, cerebrospinal meningitis, purulent meningitis, inflammation of the soft cerebral coverings,* is an acute, sometimes epidemic disease of a heteroinfectious character, consisting of inflammation of the pia mater and marked by an irregular clinical course.

It is not uncommon in systematic works to divide leptomeningitis into numerous varieties, such as simple, serous, purulent, primary, secondary, idiopathic, epidemic, tubercular, basilar, etc. From an etiological and anatomical standpoint this is not required, but epidemic cerebrospinal meningitis will, in the general description, receive such special remark as seems required. Tubercular meningitis, however, is clinically, etiologically, and anatomically a distinct disease, and will be accorded a separate section.

Etiology.—The pial structures can be invaded in but two ways: by traumatic or destructive lesions of the bony and fibrous envelopes of the brain on the one hand, or through the vascular supply on the other. To the first group belong those cases of meningitis arising from direct inoculation, as in cranial fracture and septic extension from neighboring foci in the scalp, face, cranial bones, the middle ear, the mastoid cells, the nasal fossæ, antra, and sinuses, and from the orbit and pharynx. To the second group belong the larger number, which, formerly called idiopathic, are now known to depend on microbic infection. The exact infection path is often problematical. Ortman pointed out the presence of coryza in a large proportion of the cases, and supposed that the infection reached the pial space through the lymph-channels of the nasal vault, which are continuous with those of the brain.

In the epidemic form Scherer[1] again calls attention to the severe nasal catarrh at the commencement of the attack. Weigerts in eighteen cases found purulent nasal catarrh, and demonstrated Weichselbaum's diplococcus intracellularis meningitidis in the secretions. In fifty persons not suffering from the disease he found this diplococcus in two instances and supposes that it is inhaled, taken up by the leukocytes, and by them carried into the lymph-spaces of the brain through the nasal vault. Flexner and Barker[2] emphasize the probability of the infection atrium being in the intestinal tract. The close relation of pneumonia to meningitis has for a long time pointed to the lungs as an invasion route. As a clinical fact, any infection at any near or remote point may induce meningeal inflammation by way of the vascular system.

Bacteriologically, a case of acute meningitis may present one or many organisms. Those most frequently found alone are Weichselbaum's diplococcus, the pneumococcus, the streptococcus, the typhoid bacillus,[3] and the bacillus coli. In association with them, and perhaps often as a secondary infection, are found the staphylococcus aureus and albus and various indeterminate streptococci and bacilli. The most common of all and the most significant are the pneumococcus and the diplococcus intracellularis. These microörganisms are found in the meningeal fluid, but may, as in a general pneumococcic infection, be widely distributed throughout the body. Lately the diplococcus of Weichselbaum has been demonstrated in the pus of arthritis, occurring in a case of meningitis, and also in the blood.[4] Osler, in the Cavendish lectures for 1899, made the following practical classification:

ACUTE LEPTOMENINGITIS.	Primary.	1. Of cerebrospinal fever.	(*a*) Sporadic. (*b*) Epidemic.	**Diplococcus intracellularis.**
		2. Pneumococcic.	Meninges alone involved or in a general pneumococcus infection.	**Pneumococcus.**
	Secondary.	1. Tuberculous	. . . .	**Bacillus tuberculosis.**
		2. Pneumococcic.	(*a*) Secondary to pneumonia, endocarditis, etc. (*b*) Secondary to disease or injury of cranium or its fossa.	**Pneumococcus.**
		3. Pyogenic.	(*a*) Following local disease of cranium or a local infection elsewhere. (*b*) Terminal infection in various chronic maladies.	**Various forms of staphylococci and streptococci.**
		4. Miscellaneous acute infections.	In typhoid fever, influenza, diphtheria, gonorrhea, anthrax, actinomycosis, and other acute diseases.	**Typhoid bacillus, influenza bacillus, diphtheria bacillus, gonococcus, etc.**

It will have been noticed that leptomeningitis is found in frequent association with the other infectious diseases. Curschmann has noted it in small-pox and scarlatina, both with and without purulent otitis. It is not rare after typhoid. It is common with pneumonia. All pyemias, whatever their source, have their recorded cases. Acute articular rheumatism is frequently attended by meningeal symptoms which are customarily attributed to rheumatic inflammation of the serous brain-envelope, but Ball,[1] in at least 3 out of 69 such cases, found a purulent effusion, and serous exudation was present in 35. Mumps are often associated with meningitis and epidemics of both have been concurrently noted. The same is true of coryza, which, like parotitis, is sometimes undoubtedly due to the lancinate micrococcus. A most marked relationship exists between pneumonia, influenza, and meningitis. They may follow one another in a given patient. They are apparently interchangeable in a grippal epidemic, and present remarkable bacterial analogies. In pneumonia of the apex a meningitic disturbance sometimes arises that is not marked anatomically by any evidence of inflammation. Cultures in such cases have also been negative.[2] The clinical picture, however, is that of acute meningitis, and further investigation may yield positive findings. The so-called acute serous meningitis is generally secondary to some pyemic condition, and in some instances the clear meningeal fluid has contained streptococci.[3] Insolation is undoubtedly at times attended by a meningeal congestion that may develop into active inflammation with a tendency to chronic changes.

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[1] "Centralbl. f. Bakt. u. Parasitenkunde," April, 1895.

[2] "Am. Jour. Med. Sci.," Feb., 1894.

[3] Daddi, "Lo Sperimentale," July, 1894. Ohlmacher, "Jour. Am. Med. Assn.," Aug. 28, 1897.

[4] Gynn, "Phila. Med. Jour.," vol. ii, No. 24.

Pathological Anatomy.—The pathological changes in *the meninges* are more or less circumscribed when due to infection by extension, and are then limited to the neighborhood of the primary lesion. On the other hand, infection by way of the circulation gives rise to a generalized meningitis which is most intense at the vertex of the hemispheres and may completely spare the basal region. The dura mater, except at the site of bone disease or similar infection center, is practically intact and is readily removed. The pia presents a roughened, marbleized appearance. The vessels are engorged with blood and the pial spaces are filled with a serous, milky, or purulent exudate, which follows the vascular courses, fills the sulci, and, if sufficiently abundant, unbrokenly covers the convolutions. At other times the exudate appears in discrete patches, which are for the most part found at the basilar outlets of the cranial nerves and vessels. Extensions sometimes accompany the auditory nerve into the internal meatus or follow the optic nerve into the orbit. The seropurulent deposit is more or less fibrinous, and the serous exudate sometimes is filled with flaky masses, which escape with it when the skull is opened. Ordinarily the inflammation follows the choroid plexuses into the *ventricles,* which are oftentimes dilated by the increased turbid, flocculent, pial fluid.

The cerebral *cortex,* in cases of short duration and in the so-called serous form, may show little more than the evidence of increased vascularity. In severe and protracted cases minute hemorrhages are common, both in the pial spaces and the brain-substance. The perivascular sheaths are blocked with exudate, and the cortex is edematous, infiltrated with pus, and adherent to the pia, which can not be separated from it without stripping off the gyral substance. Thrombotic softening and abscess formation may follow.

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[1] "Thèse de Paris," 1869.

[2] Bergé, Claisse, "Traité de Médecine," vol. vi, p. 529, Paris, 1894.

[3] Nobecourt and Delestre, "Annales de Med. et de Chir. Infantiles," April 15, 1900.

The *cord* is affected in about one-third of the cases. Its meningeal and deeper conditions are similar to those of the cerebrum. The posterior roots seem especially vulnerable, and present marked inflammatory and degenerative changes of their hyaline and axis-cylinder elements. The exudate is usually thicker on the posterior surface of the cord, probably from the usual dorsal position of the patient. This may also account for the preponderance of changes in the posterior part of the cord.

From the gross appearance it is evident in severe cases that meningitis is attended by a certain degree of *cerebritis.* The anatomy of the meninges explains this. The histological changes consist of capillary and vascular dilatation in the pia and an active diapedesis into the perivascular sheaths. These are dilated and crowded with leukocytes and purulent elements. The neurogliar cells and network of the cortex show some proliferation. The *bacteriology* has already been indicated.

The *body organs* show the varying lesions of the primary disease, such as typhoid, pneumonia, infectious endocarditis, or local septic processes. Splenic enlargement may alone mark the infectiousness of the disease.

Symptoms.—The *incubation period* of meningitis is an indefinite one. In some epidemics it has appeared to be somewhat less than a week. In the fulminant cases the severest manifestations of the disease are present almost in a moment, and death may occur in twenty-four hours. Usually there is an *invasion period* of several days or weeks, attended by malaise, discomfort, slight feverishness, and headache. More pronounced disturbance then ensues, and we have a varying *period of excitement,* followed by one of depression, stupor, coma, and death.

The prodromal *headache* becomes severe, continuous, and of all the symptoms is the most constant and significant. It is particularly violent and unmanageable. The patient constantly complains of it, and when stupor or coma has supervened, by holding the head in the hands and by moans and facial expression, he still indicates its often overmastering presence. In children it gives rise to the sharp cephalic cry that punctuates their stuporous state at frequent intervals. Ordinarily it is referred to the occiput or vertex, but is often diffuse.

Delirium is common in children and frequent in adults. A mental fogginess is often early noticeable. The patient, racked by the cephalalgia, seems irresponsive, unimpressionable, and is hazy in his replies. The delirium is of a low grade generally, but may be wild and frantic or suggestive of the busy delirium of alcoholism and typhoid. *Vomiting* of a projectile character is rarely absent in children, but is less common in adults. The stomach seems simply intolerant and rejects without nausea the unchanged ingesta. It may be an early symptom. At the same time the tongue may be quite clean; later it is often thickly furred and suggestive of typhoid. The *bowels* are usually constipated and the *abdomen* retracted.

Convulsions in the early stages, particularly in children, often occur. They are general in character and of protracted duration. When the convexity is invaded, they may later present a limited distribution, one side of the body, the face, or a single extremity being alone involved.

Almost invariably there is more or less muscular *rigidity*. In most cases this is marked at the neck by a tendency to retraction that is highly significant. At first the patient complains of a feeling of nuchal stiffness and soreness, and finds slight relief in resting the head on a chair-back or over a firm pillow. In the comatose condition the head is often strongly retracted and the occiput drawn well between the shoulders. When less marked, an attempt to passively bring the head forward will provoke distress and resistance. A similar rigidity rarely invades the muscles of the lower jaw, producing slight trismus. It may involve the extremities, and when the meningitis has attacked the spine the trunk is often held rigidly in a position of dorsal extension. Kernig and Bull [1] have described a peculiar rigidity in the lower limbs. If the patient is placed in a chair he is unable to extend the knees owing to the contracture, which disappears when the thigh is straightened on the trunk. According to Herrick, Osler, and others, this sign is practically constant in this disease. It appears early and is certainly valuable. *Muscular weakness* is usually present and may be more or less localized.

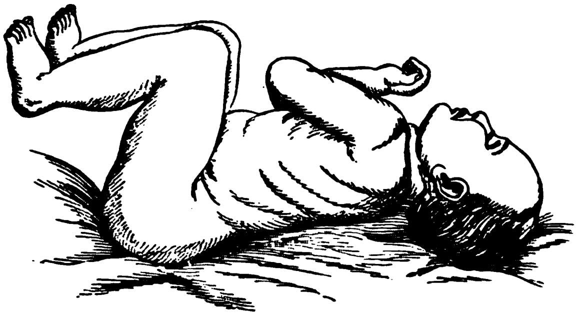

Fig. 27.—Attitude of child with cerebrospinal meningitis (Smith).

The *cranial nerves* in most cases sooner or later show invasion and furnish valuable diagnostic symptoms. The olfactory nerves are seldom disturbed, but occasionally the patient complains of olfactory sensitiveness. Photophobia is a common symptom. The *optic nerve* is irritated by extension of the inflammation down its sheath. After a few days haziness of the disc and enlarged vessels are often seen ophthalmoscopically. The disc may be markedly obscured. Retinal hemorrhages, papillitis, and subsequent atrophy and blindness are occasionally encountered. Choroiditis, or panophthalmitis, is sometimes present and may cause great injury to the eye or result in complete blindness. The *third nerve* is almost always affected. When marked strabismus does not demonstrate it at a glance, by having the patient turn the eyes in various directions a lack of conjugate action becomes apparent. Questioning may develop the history of visual uncertainties—haziness or transient diplopia. The *pupils* also furnish important signs. Early in the attack there is a tendency to miosis, which later is replaced by pupillary dilatation. The reflex to light is lost or greatly reduced in amplitude or activity. The pupils may be perfectly immovable. They are often unequal. The *facial nerve* is exceptionally paretic, allowing the face to deviate to the opposite side. At the same time the *auditory nerve* is implicated through its association with the facial nerve within the bony walls of the internal meatus. The loss of hearing that sometimes follows cerebral meningitis is due principally to an extension of the inflammation to the labyrinth, and only exceptionally to destruction of the auditory nerve-trunk. Irritation of the auditory apparatus is apparent in the early stages of the attack. All sudden or loud noises greatly distress the patient. Implication of the *hypoglossus* in rare cases causes deviation of the tongue. Probably all other cranial nerves are similarly affected. Doubtless the common respiratory and cardiac disturbances are, at least sometimes, attributable to injury of the pneumogastric.

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[1] "Berlin. klin. Wochens.," 1894-1895.

In addition to the hyperesthesia of the special senses much *tenderness* is customarily found over the spine and limbs. The back of the neck is especially sensitive, and firm pressure of the muscular masses is usually resented. The entire head is more or less sensitive to deep pressure, and pain is provoked by gentle percussion over the skull. The paretic and sensory disturbances may be unilateral or monoplegic in distribution. The location of the disease causes these variations.

Cerebral meningitis presents no characteristic *temperature* curve. It may be ushered in by a chill and an elevation of the body-heat to 104° or higher. It may show a very low, even a subnormal, temperature at first, and terminate with a high range. The evening rise may default. Fickleness is the rule and, in a way, diagnostic.

The *pulse* shows variation in two directions. During the early stage of excitement it is likely to be full, active, and greatly accelerated. Not uncommonly, however, the physician is surprised to find a sluggish pulse of 40 or 60 and the temperature mounting above 100°. This *dissociation of pulse and temperature* is usually manifest sooner or later in meningitis, and furnishes a sign of capital importance. Toward the fatal end the temperature bounds upward, attended by a pulse of great, almost uncountable, rapidity.

Vasomotor signs are not lacking. *Herpes labialis* is as common as in pneumonia and as significant. If the finger-nail be drawn across the integument of the abdomen or elsewhere, it is followed slowly by a congested red streak that persists for many minutes. Trousseau laid much stress on the phenomenon, which he called the *tache cérébrale*, and it is of some significance. Taken alone it is of no importance, as it is common to many conditions. The application of mild irritants or gentle heat is likely to be followed by vesiculation and sloughing. *Urticaria* is often present. A red *macular eruption* which gives to cerebral meningitis its sometime name of spotted fever is of infrequent occurrence. It appears mainly on the abdomen and trunk, and may be mistaken for the petechial marking of typhoid fever. It is more like roseola.

Early in the attack the *respiration* may be quickened. Later, in the stupor and coma, it is often slow and irregular. The Cheyne-Stokes variety is often observed, and is of very serious though not absolutely of fatal import. The ordinary relations of respiration to the temperature and pulse may be, and often are, disturbed.

The *urine* shows the general febrile disturbance, and is scanty, high-colored, and heavy. Sometimes albumin and sugar are found in quantities varying from a trace to large amounts. They are significant of irritation of the medullary centers in the floor of the fourth ventricle.

The *tendon-reflexes* may or may not be much modified. Sometimes they are exaggerated, more often decreased. It is not uncommon to find them diminishing as the depressed stage comes on. Their obliteration after the disease has lasted a week or ten days in protracted cases is often noticed. They may be more disturbed on one side than on the other. The toe-sign is frequently encountered.

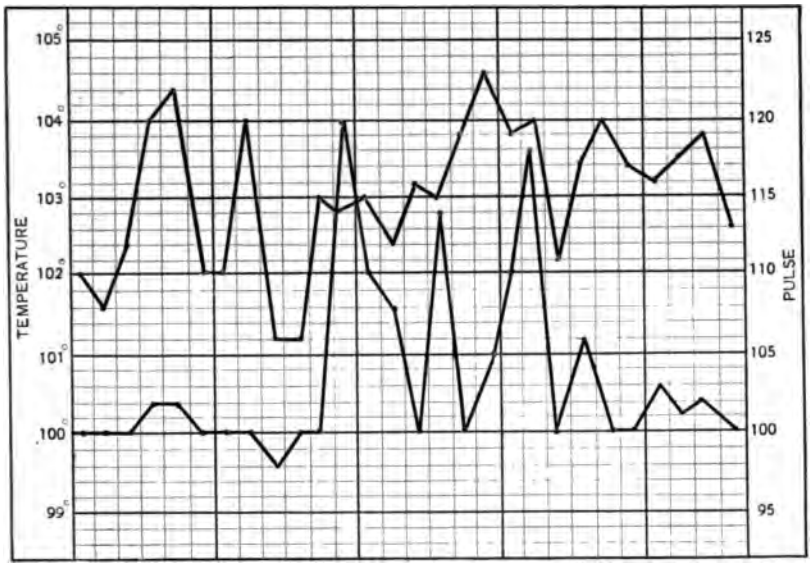

Fig. 28.—Partial temperature- and pulse-curves in a case of meningitis of the convexity, showing dissociation. Temperature, upper line; pulse, lower line. Record taken every four hours, commencing at midnight.

Course.—No two cases present parallel conditions as to course, symptoms, or intensity. A fulminant case may end fatally in a day. Protracted cases extend over several weeks and some are clearly subacute. An ordinary non-epidemic case presents perhaps a week of malaise, a week of excitement, and a week of depression, which usually terminates in deepening coma and death by cardiac failure. At any time, even when the patient seems to be *in extremis*, the symptoms may clear away and convalescence follow. A remission of symptoms may give false hope, to be destroyed by the return of the serious features of the disease in all their intensity. When the basilar region is principally affected, the course of the disease is likely to be cut short by bulbar invasion. The convexity cases are of longer duration and the patient thus affected is sometimes gradually worn out by the pain, delirium, and fever.

During the period of excitement of functions, vomiting, delirium, vertigo, high temperature, muscular rigidity, spasms, convulsions, indeterminate headaches, and hyperesthesia are the prominent features. They are gradually replaced by increasing apathy, stupor, and coma, in which the patient lies inert. He may be nearly relaxed or lie with retracted head, marked squint, and rigid limbs, passing urine and feces under him, and responding to strong stimulation slightly or not at all. The rapid respiration of the first period is replaced by the slow or irregular breathing that indicates increased intracranial pressure or pneumogastric irritation. Finally, deglutition is impaired, respiration becomes stertorous, and the patient slowly sinks or may expire in a convulsion.

Various epidemics have shown numerous features of similarity among the cases that have marked their progress. At first the fulminant cases have been common. At the end those of less and less severity are met with.

Diagnosis in the very early stage is difficult. When the disease is fully developed, and in epidemic conditions, a mistake would be almost impossible. No one symptom is constant, and it is a disease of protean aspects. The symptoms vary as the base or convexity is involved. Meningitis of the convexity is marked by excessive delirium, high temperature, convulsions, localized spasm, dysesthesia, and monoplegias. The cranial nerves are not involved, and ocular symptoms are slight or entirely wanting.

When the base is affected, the temperature often has a low range or is subnormal. Cranial palsies are common, retraction of the head is nearly always present, and the dissociation of pulse, temperature, and respiration is marked. Optic neuritis declares the basilar location or extension of the disease. As a rule, infection by the blood-channels results primarily in meningitis of the convexity. In rare instances the meningitis is confined to the ventricular area and the choroid appendages, but no distinctive symptoms indicate this limitation. The diagnostic value of the headache depends upon its duration, intensity, and particularly upon its persistence after delirium has appeared or coma has ensued. The tenderness of the head and the pain on pressure are of some value. Vomiting is only significant when of the projectile character, unattended by gastric disturbance and fermentative changes. The temperature lends aid when out of keeping with the pulse-rate and respiration rhythm. The tenderness in the limbs, the rigidity of the neck, the contractures at the knee in the sitting attitude, are highly suggestive. Apathy, drowsiness, and mental obscuration in themselves should suggest the disease. The grouping of several of these symptoms would justify a tentative diagnosis, which the appearance of squint, convulsions, delirium, optic neuritis, Kernig's sign, or the vasomotor symptoms would confirm. Gowers states that in suspected cases the too free vesiculation of the skin under heat or irritation would lend support to the diagnosis of meningitis. Lumbar puncture will also assist.

Differentially, the diagnosis is often much complicated by the association of meningitis with other affections which may overshadow it. As it occurs in the course of pneumonia, typhoid fever, cranial injuries, septic invasion, and pyemias, the original disease process may entirely absorb the practitioner's attention. From typhoid, Hirt[1] says, it is sometimes indistinguishable, presenting a similar temperature curve, splenic enlargement, rose spots, and the typhoid stool. Widal's reaction is a helpful but not absolute sign of typhoid. The presence of pseudomeningeal symptoms in pneumonia of the apices has been mentioned, and in pneumonia generally meningitis can only be determined by basilar symptoms. Uremia should be excluded by a thorough urinalysis. An attack of delirium tremens may be easily mistaken for meningitis, with which it is not rarely complicated. Single or double acute otitis in children, marked by constant pain and vomiting, delirium, and deafness, is usually confounded with meningitis. If the facial nerve escape, the deafness would point to labyrinthian disease and serve to exclude meningitis. Hysteria is sometimes mistaken for meningitis. The emotional and mental features, the normal temperature, breathing, and pulse, and certainly the stigmata of hysteria should differentiate it. In children the onset of nearly every febrile disturbance has been confounded with meningitis. Here reliance must be placed on the characteristic temperature curves, eruptions, and clinical antecedents. The character of the infection can usually be determined by spinal puncture and appropriate bacteriological tests.

Prognosis.—In expressing an opinion as to the outcome of a given case, the physician has three positive statements to make. First, the result is absolutely uncertain until death occurs or convalescence is firmly established; second, the probability of a fatal termination is always pronounced; third, some trace of the disease is likely to remain permanently. The disease is full of surprises. The writer has seen a patient in deepest coma for hours, with convergent squint of the most marked variety, retracted head and abdomen, convulsed limbs, Cheyne-Stokes respiration, large quantities of sugar in the urine, and a pulse of 32, entirely recover. Again, cases that seem trifling or on the high road to recovery suddenly become worse and die. Purulent cases are practically always fatal. If a purulent focus within the skull, nose, ear, or throat is found, it renders the outlook extremely grave. A meningitis following pneumonia is practically fatal, but Stoeltzner[2] reports a recovery where spinal puncture showed both pus and the pneumococcus. The cerebrospinal form runs a more favorable course, the mortality varying from about thirty to sixty per cent. in various epidemics. Partial recoveries, however, greatly outnumber cures. The extension of inflammation to the labyrinth produces deafness, which in young children may lead to deaf-mutism. Optic neuritis is followed by dimness of sight or complete blindness. Local meningeal thickenings and cerebritis portend monoplegias, epileptoid convulsions, and mental defect. Though the disease is not one of proved contagiousness, separation of the patient from the other members of the family may be insisted upon with propriety. The tubercular variety is almost invariably fatal.

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[1] Hirt, "Diseases of the Nervous System," New York, 1893.

[2] "Berlin. klin. Wochens.," April 19, 1897.

Treatment.—The first consideration in the management of a case is the removal, if possible, of the infection atrium. Cranial suppuration and injuries demand immediate surgical attention. Optic, nasal, pharyngeal, intestinal, and pelvic cavities should be rendered aseptic if their organs or contents fall under suspicion. Intestinal antisepsis is always in order. To this end free catharsis and the administration of antifermentatives are indicated as well as by the usual constipation. Minute doses of calomel, $\frac{1}{10}$ of a grain, repeated every half hour or hour until active results are obtained, can be highly recommended for this purpose. In addition, the mercurial has a widely diffused microbicidal value.

From the first the patients should be kept in a cool, quiet, moderately darkened room, and all annoyances to which their hyperesthetic senses make them especially responsive should be prevented. Extreme delirium will often require a sedative. In the presence of the cephalalgia bromids, chloral, Indian hemp, and other ordinary means will practically always fail. The coal-tar derivatives are equally valueless, and nothing but morphin will give relief. Its hypodermatic administration is by all odds to be preferred except in infants. If high temperature be present, antipyretic drugs must be employed with great circumspection. They often have no effect, and their depressing action sometimes constitutes a real danger in a disease marked by symptoms of disturbed cardiac and respiratory innervation. Tepid to cool baths with cold affusions to the head, sponging, and the pack may be used to better advantage. Hot baths are decidedly valuable. They often allay the delirium, reduce the temperature, and clear up the clouded mind. The use of an ice-bag or ice-coil to the head frequently gives comfort to the patient and renders the head-pain bearable. Counterirritation along the spine is a measure of doubtful utility, and always attended by the danger of setting up serious ulceration or actual sloughing.

In the depressed period sedatives are no longer required, and stimulants, like strychnin and whisky, will often need to be sharply pushed to meet the failing action of the heart or lungs. When coma has appeared, it may often be broken and sometimes happily ended by the application of a fly-blister to the nape of the neck. It should be sufficiently large,—two by six inches for an adult,—and extend from one mastoid to the other. If this is not efficacious or the coma again develop, a drastic cathartic—one to five drops of croton oil in emulsion—may render similar service. By recurring in turn to the blister, cathartic, and baths, with cool affusions to the head, no doubt the progress of the disease may be often hindered. Unfortunately, it usually again takes up its course, but occasionally such measures seem to definitely check the malady and recovery slowly follows. Aufrecht[1] recommends full hot baths of about 100° F. for ten minutes. Their apparent effect sometimes is to reduce temperature, diminish the headache, accelerate the pulse, and induce sleep. As many as eight a day have been given with excellent results by Woroschilsky.[2]

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[1] "Therap. Monatshefte," Aug., 1894.

[2] *Ibid.*, Feb., 1895.

The use of large doses of iodids with the expectation of causing resorption of the effusion usually only results in distressing the patient's stomach, reducing his strength, and increasing the vomiting. Shaving the head is rarely called for, but a heavy mass of hair may usually be removed with advantage. Mercurial inunctions seem at times to lend help even in the absence of syphilitic taint. It is immaterial whether they be applied to the scalp or to the limbs, so far as the effect is concerned, but the non-hairy parts are more convenient and less irritable. Credé's colloid of silver ointment and iodoform ointment in large doses applied to the head have advocates, and may be properly employed. Good results have been claimed for subcutaneous injection of the bichlorid of mercury in doses of $\frac{1}{10}$ of a grain several times a day in children. There is some reason to hope that an antitoxin may be developed to combat the epidemic form.

The most important items in the treatment of meningitis are the nursing and nutrition of the patient. A careful, intelligent, preferably trained, nurse who will exclude visitors and members of the family and prevent disturbing sounds, bright lights, and all annoyances, is the salvation of some cases. Thus only can constant watchfulness of pulse, respiration, and temperature, which may show extreme and critical variations in an hour, be provided and emergencies met as they arise. Nourishment in small quantities can usually be administered frequently. If the cerebral vomiting persist, morphin is practically the only measure we can rely upon to check it. In the stuporous states rectal alimentation will be serviceable, or the nasal stomach-tube in skilled hands may be employed. If deglutition is difficult, one of these is imperatively demanded. Large quantities of soups, custards, whey, junket, beef-juice, and eggs may be given with advantage from first to last. The bowels must be active.

The question of *trephining* the cranium and draining the meninges and ventricles has received favorable consideration in those cases where deepening coma and failing circulation point to increasing intracranial pressure. The results thus obtained, however, are not encouraging.

Quincke, in 1891, suggested that the intracranial pressure could be easily relieved by puncturing the dural sheath of the lumbar cord with a hollow needle. The procedure has since been widely adopted. Furbringer [1] used the *lumbar puncture* one hundred times in eighty-six cases. In many of his non-tubercular cases sugar was found in the spinal fluid, but only exceptionally in tubercular meningitis. The presence of pus or tubercle bacilli or other organisms in many instances completed the diagnosis. In several cases in children a sinking of the fontanel showed that the intracranial pressure was reduced. A. Fraenkel [2] has noticed the optic papillitis diminish and improvement in other symptoms after the puncture. The writer has observed a pulse of 160 drop twenty beats in an adult upon the removal of two ounces of fluid. As a means of diagnosis it has a precise value when positive findings are furnished, but therapeutically has not accomplished much. In principle it seems seductively correct, and in a disease of such fatal character may be employed in suitable cases with proper surroundings when pressure symptoms are marked. The operation is a simple one. It is best performed with the patient in a sitting position, the back bent well forward, thereby opening the spaces between the lumbar spines. Under full asepsis a hollow needle of one and one-half millimeters is introduced from without inward, about an inch from the middle line between the third and fourth lumbar spines. This level is below the cord. The fluid at first usually flows rapidly, but later drop by drop and shows the influence of respiratory pressure. The amount withdrawn has varied from a few drops to several ounces. The puncture may be repeated as required.

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[1] "Berlin. klin. Wochens." April 1, 1895.

[2] *Ibid.*

One danger of lumbar puncture is pointed out by Jacoby,[1] who suggests that where the meningitic process is confined to the cerebrum draining by the lumbar puncture may carry the infection down the cord, and thereby extend the disease. He also shows conclusively that it is impossible from the lumbar extremity to force fluids of a therapeutic character into the arachnoid spaces above the cervical region, unless puncture is also made into the ventricular space of the brain, when the fluid passes very readily from one end to the other of the cerebrospinal meninges. He advocates such flushing of the cerebrospinal axis by means of both the lumbar puncture and a small trephine opening with drainage from the lateral ventricle.

In recovered cases treatment is directed toward building up the general health. Inflammatory thickening and remaining effusion may perhaps be benefited by iodid of potassium and mild mercurial courses. Weakened or paralyzed extremities should receive careful electrical and massage treatment as soon as the active febrile stage subsides. All severe physical and mental fatigue should be avoided for a long period. Exposure to the sun and the use of stimulants will be found especially detrimental.

Chronic Leptomeningitis.—Leptomeningitis, as already mentioned, is occasionally subacute. This form may become chronic. As the result of alcoholic excesses, syphilis, and insolation, sometimes a low grade of chronic leptomeningitis is developed. Post-mortem examination frequently reveals such a condition which may have been unsuspected during life. Among the chronic insane it is a very common finding after death. Clinically, except in the syphilitic form, its manifestations are very uncertain and obscure. Stiffness of the neck and persistent slight headache, both marked by exacerbations, with some tenderness over the skull, may be complained of. Undue optic and auditory sensitiveness usually accompany the more severe periods of headache. All causes of cerebral congestion, such as stooping and muscular or mental exertion, cause distress and intensify the headache.

The alcoholic cases are the least well marked. A low grade of optic neuritis may be present, and usually disappears if the alcohol is withdrawn. Slight mental cloudiness or delirium, due to the specific action of the poison on the brain-tissue, is frequently observed. Multiple neuritis and other indications of alcoholism are usually present, and often quite overshadow the meningeal symptoms.

The syphilitic inflammation is usually circumscribed, and gives rise to local symptoms. It will be fully considered under the head of Syphilis of the Nervous System, Part VI.

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[1] "New York Medical Journal," 1895.

A chronic infantile meningitis has been described in which the meningeal involvement is confined to the posterior basal fossæ of the skull. It may last from a month to several years, and terminate in death or recovery. Its onset is sometimes sudden, and has been shown in certain cases to depend on hemorrhage about the medulla and pons. In other cases it is of gradual development. Palsies are rare; retraction of the head and strabismus are common. Through damming up of the ventricular outlet, hydrocephalus may be induced.

CHAPTER III.

TUBERCULAR MENINGITIS.

Tubercular leptomeningitis, *basilar meningitis, acute hydrocephalus*, is a local manifestation of the action of Koch's bacillus, usually most intense at the base of the brain, commonly attended by distention of the ventricles, almost invariably if not always secondary, and practically always fatal.

Etiology.—The pathological process in this form of meningitis is clear and definite. The bacillus of tuberculosis carried to the meninges of the brain by the vascular system finds in the pial fluid and the perivascular spaces a suitable pabulum and an excellent breeding ground. In the great majority of cases a primary focus of tubercular infection is found in other organs. The absolute exclusion of such original disease is practically impossible in the few remaining cases where it is not readily discovered. It has been suggested that infection might reach the cranial contents by way of the nasal vault and cribriform openings. This can not be denied, but seems improbable and lacks confirmation. Though tubercular infection of the soft brain-coverings may occur at any *age*, the years of life between two and ten show an overwhelming proportion thus affected. Before the age of six months and in advanced years it is practically unknown. It gradually diminishes after the age of ten and is infrequent after thirty-five. The *other etiological factors* are those that are common to all tubercular processes. Heredity, both tubercular and neurotic, has a significance. Urban populations, conditions of crowding, winter and spring (the seasons of exposure and poor ventilation), depressing influences, deprivation, and tubercular contamination are included in the list of predisposing and exciting causes. Traumatism may induce a tubercular meningitis perhaps by reducing the resistive factor in the tissues that stays the bacillary invasion. At any rate, it is a common observation that spinal and head injuries in themselves rather trifling, are followed by the disease in numerous instances.

Pathological Anatomy.—Upon opening the skull and reflecting the dura, hardly any inflammatory action is, as a rule, to be seen. The arachnopia is sometimes a little lacking in luster along the course of the main cerebral arteries. At the base, however, the morbid picture is comparatively uniform and striking. At the arterial circle of Willis, extending along the basilar furrows, between the peduncles and the pons, covering the interpeduncular space, and especially marked in the Sylvian fissures, is a thick, almost gummy exudate, dotted with small masses of a dirty whitish color. These are tubercles in various stages of development or degeneration. They frequently dot the pia on the lateral aspects of the brain and sometimes reach to the vertex. They are always grouped near the blood-channels and vascular spaces, through which evidently the infecting organism reaches the meninges.

The *exudate*, often of a clear, jelly-like consistence, is frequently turbid, grayish-yellow, and not rarely purulent or even greenish. It is found in greatest quantities at the base, but ascends with the cerebral vessels along the pial spaces, filling the sulci and interlobular grooves. It travels down the perivascular sheaths into the cortical substance, and invades the ventricles through the transverse fissure. The inflammation is here propagated to the ventricular ependymal lining, resulting in a great increase of fluid, with dilatation of the ventricles and a condition which early gave to this disease the distinctive name of *acute hydrocephalus*. This feature is seldom wanting and may, by pressure, cause marked flattening of the convolutions.

The granular *tubercles*, usually visible at a glance, sometimes require a little search. If the pia be stripped off and floated in a little clear water, the tubercles can be much more readily distinguished. They are grouped about the arterioles or disseminated along the larger vessels in patches and small masses, which may rarely unite to form a continuous covering for wide areas of the brain-surface. They present the variations which mark tubercles elsewhere, depending upon their age and development or disintegration. Microscopically, they are often found to occlude the perivascular spaces, giving rise at times to small softenings and hemorrhages, which occasionally attain sufficient proportions to explain the focal symptoms that may have been clinically manifest. These infarcts are usually found in the basal ganglia and the cerebral peduncles. The perivascular extension of the inflammation determines more or less *cerebritis*, and when the pia is removed it often decorticates the brain in consequence of the soft adhesions that have formed. Infrequently there are slight adhesions between the pia and dura.

In a large proportion of cases of tubercular meningitis the *spinal cord* is also invaded. The meninges, meningeal vessels, and the cord itself present features analogous to those found within the cranium. The exudate is most marked in the anterior and posterior grooves of the cord, and the meningeal changes are always greater on the dorsal surface.

Tubercular meningitis presents *varieties* in which the pathological findings are most marked at the vertex. This is so rare that the disease was formerly known and described simply as basilar meningitis. Occasionally a circumscribed tubercle or a tubercular mass gives rise to symptoms of, and constitutes, an intracranial tumor. Again, the meningeal involvement is only a part of disseminated miliary tuberculosis, appearing at once in the head, lungs, intestines, peritoneum, and abdominal parenchymatous organs. In these cases, while all the pial structures are the seat of granular tubercles, they are most profuse along the vascular routes. Ordinarily, they are not attended with much exudation and brain symptoms may be quite lacking during life.

Bacteriologically the tubercle bacillus of Koch is always found, and usually is present alone. Secondary mixed infections are extremely rare and purely adventitious. A case has been met in which the pneumococcus was also present. Primary tubercular processes in other organs, as in the serous sacs, the lungs, the intestines, the mesentery, the mediastinum, or the genito-urinary tract, are commonly found. A single caseous mediastinal gland has served as the starting-point for the meningeal infection.

Symptoms.—The *onset* of tubercular meningitis is insidious and its course protracted. Cases, however, manifest wide variations. The typical cases occur in childhood. Those occurring later in life are usually preceded by well-marked tubercular disease to which the meningeal complication is added in a natural order. Children, on the other hand, frequently present an antecedent appearance of good health, the primary focus of disease having been so insignificant as to produce no notable symptoms. They become peevish, fretful, and out of sorts. Appetite and sleep are disturbed. After a week or two of prodromal malaise with perhaps a little headache, an occasional vomiting spell, and slight febrile disturbance, they are noticed to be apathetic, then distinctly drowsy, and later stuporous. If disturbed, they complain of headache or manifest discomfort and may vomit. The triad of symptoms—*headache*, *vomiting*, and *constipation*—following a more or less protracted period of malaise, which may rarely extend over months, marked by general physical deterioration and often by great loss of flesh, has serious significance. The stuporous repose is often pierced, but not broken, by a sharp cry of pain, the *hydrocephalic cry*, which in some cases is frequent and distressing. It is apparently due to the head-pain. There is retraction of the head with more or less *rigidity* of the neck, and the entire spine may be fixed. Slight opisthotonos is common. The thighs are flexed on the trunk, the legs on the thighs, the abdomen becomes more and more retracted, and finally presents the scaphoid or boat-shaped hollowing that is classical. The masticatory muscles sometimes are similarly stiffened, and the Kernig symptom of rigidity of the knee when the hip-joints are flexed is usually present.

The *tendon-reflexes*, sometimes indistinct, are often increased at first and gradually diminish as the depression and stupor develop. The *vomiting* continues at intervals and is of the cerebral type, unattended by evidence of gastric disturbance. The *temperature*, though fickle, usually shows an evening elevation. It is rarely extremely high until the fatal termination of the disease, when it attains 103° to 105° F., or even more, but from 101° to 102.5° is frequently seen. Occasionally it drops below the normal and may show considerable variation in the course of an hour.

The *respiration* is not notably disturbed until stuporous or comatose conditions obtain, when it is irregular, sighing, slowed, and of the

Cheyne-Stokes variety; but Simon [1] asserts that from the first there is a lack of harmony in the respiratory movements of chest and diaphragm. The *pulse* then becomes much altered. It is slowed, often irregular, and just before death becomes uncountably frequent. It is in tubercular meningitis especially that the *dissociation* of pulse, temperature, and respiration is found. Its diagnostic value is great. Thus, when the temperature exceeds 100°, the pulse may show a subnormal rate, and the respiration be slow or rapid.

As the lesion is usually basilar, involvement of *cranial nerves* is the rule. *Sight* is often dimmed, and the ophthalmoscope demonstrates changes in the fundus-picture in a majority of cases. Simple hyperemia, marked congestion, and papillitis may be expected. Of pathognomonic importance is the not infrequent presence of tubercles in the retinal or choroid tunics. Very early there is disturbance of the *third nerve* in the form of pupillary inactivity and a tendency to miosis, which later gives way to wide dilatation. Squires [2] describes a rhythmical dilatation and contraction of the pupils caused by extending and flexing the head. As the head is bent backward the pupils slowly dilate and again contract when the head is brought forward, the pupillary variation being proportionate to the amount of flexion and extension. Strabismic deviations of the eyes, or fixity of the globes, should be carefully sought for. In older patients diplopia is frequently noted. A strong convergent upward squint, drawing the pupils almost to the inner canthus and turning the globes well up is usual in deep coma. The *facial nerve* is rather frequently affected with a corresponding paretic condition of the face. Deviation of the tongue, pharyngeal and laryngeal distress, mark the implication of the posterior members of the cranial group. The *mental state*, aside from the stupor, is one of confusion, often marked by moderate delirium. When the patient is roused or is able to respond he resents all interference. Rapid *emaciation* attends the progress of the disease. *Sphincteric control* is not often disturbed, except that in the comatose state the bladder and bowels act automatically, and their contents are unconsciously voided under the patient. Retention of urine, however, is sometimes noted and persistent; obstinate *constipation* is the rule unless intestinal tuberculosis causes diarrheal discharges.

Vasomotor disturbances are manifest in flushes and pallor, which frequently succeed each other rapidly or appear side by side on face and trunk in peculiar distribution. The *tache cérébrale* is present. The persistence and intensity of the streaks on the skin and the ease with which they are produced give some significance to a phenomenon observed in many unallied conditions.

Convulsions sometimes open the sequence of acute manifestations, but more often appear later; very rarely are they entirely absent. Due in large part to the irritation of the bulbar region, they are usually generalized and protracted. Convulsive *twitchings*, especially in the face and hands, indicate the extension of the irritation onto the lateral aspects of the brain. By localized disturbances in the motor cortex, *spasm* of a Jacksonian variety may be induced. Similarly paresis is often found, which may be localized. In cases of protracted onset, some wavering in the gait, or even marked staggering and clumsiness, are of the same significance.

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[1] "La France Méd.," March 29, 1895.

[2] "N. Y. Med. Rec.," Mar. 26, 1904.

After a variable, active period of a few days, a week, or even longer, a marked *remission* of the stupor, convulsions, vomiting, and other signs of active disease is usually noted. It is frequently followed by a period of vacillating improvement that only too often gives false hope and ill-founded security. After a number of oscillations, in some of which the patient may seem to touch the border-land of complete relief, the disease again takes up its course to a fatal termination. Such remissions may last from one month to a year,[1] and terminate without cause, or apparently as the result of a fall, shock or intercurrent slight illness. All the symptoms of depression reappear, the coma intensifies, the pressure indications increase, the temperature runs up, stertor comes on, the pulse is inordinately accelerated, and death from respiratory or cardiac failure closes the scene, often attended by convulsions, due, perhaps, to the asphyxiated blood state. Occasionally a stuporous or even comatose condition, lasting days and weeks, may precede the fatal termination. *Spinal symptoms* are common. The rigidity of the back and lower extremities, and great tenderness along the spine and over the skin, indicate spinal involvement; but the overwhelming cerebral side of the disease usually obscures the less strongly marked features of cordal extension.

Course.—A disease marked by such pronounced variations of intensity, and even of localization, necessarily presents a lack of uniformity in its course and duration. Most frequently it is subacute. Rare cases reach a fatal termination in a week or ten days after the onset of marked symptoms. On the other hand, the prodromal stage may extend over weeks and months, with remissions of all symptoms at intervals. Again, the mid-period, marked by fluctuation, may protract the disease for days and weeks. Ordinarily, the malady presents four fairly marked clinical stages: (1) The prodromal period, of indefinite length; (2) the period of irritation and excitement, varying from a few days to a week; (3) the period of oscillation, lasting about as long, and, finally, (4) the period of marked depression, attended by paralytic features, deepening coma, and death. This refers principally to the disease as it occurs in childhood. In adults it is likely to run a more varied and protracted course, and in the rare senile cases it often provokes but insignificant disturbances.

The diagnosis is avowedly difficult. In the prodromal period it is never positive. When drowsiness, headache, vomiting, and constipation are found following a week or more of malaise and petulance, the meningeal character of the trouble would be pertinently suggested. The detection of a primary tubercular process, or of tubercles in the retina, is a practical confirmation of the diagnosis. Strong hereditary tendency to tuberculosis, and more emphatically a family history of

[1] L'Hote, "Thèse de Lille," 1904.

several cases of tubercular meningitis, would raise a strong presumption, when attended by the cerebral triad, headache, vomiting, and constipation, that tubercular meningeal infection had taken place. Continued exposure to tubercular infection has some significance. The differential diagnosis from acute meningitis depends principally on the subacute onset and lower grade of intensity of all the symptoms in the tubercular variety. This is manifestly indefinite, and clinically the two forms of meningitis can not always be distinguished. Quincke's puncture furnishes a practical test. The bacillus tuberculosis is found in the spinal fluid in about four-fifths of the tubercular cases. In other cases the presence of the pneumococcus or of pus has made a differential diagnosis possible.

Prognosis.—Practically the only hopeful point in prognosis is the possibility of a mistaken diagnosis. A few undoubted cases of tubercular meningitis have recovered, and the post-tubercular lesion has been found after death from subsequent and unrelated causes. Dr. G. Futterer,[1] formerly assistant to Rindfleisch, relates a case in which tubercular meningitis had been diagnosed by Prof. Leube. Five years later calcareous tubercles were found in the spinal meninges. Henoch, Pollitzer, Freyhan, and Avanzino[2] also report cases that have a similar bearing. Their extreme rarity hardly invalidates the rule of fatality. Owing, in some cases, to the difficulty of a differential diagnosis, the physician should always maintain a margin of reserve. This is particularly important in the oscillatory period of the disease, when apparent recovery may be most deceptive.

Treatment.—The same general management and care should be employed as in acute meningitis. Actuated by the beneficial results of iodoform injections in tubercular joint-disease, in Germany inunctions of the shaven scalp with the same medicament have been warmly advocated. They may be tried. All are united on the use of calomel in small repeated doses. The mercurial not only is the best agent against the constipation, but may have some effect upon the inflammatory process within the skull. The extreme tendency to rapid emaciation suggests the most careful and persistent efforts to maintain the nutrition by every possible means. When the stomach is intolerant, rectal alimentation may be employed and cod-liver oil inunctions are useful. Digestion can sometimes be assisted by peptonizing and predigesting the food. Stimulation by whisky or a good wine is important in the later stages. During the period of excitement sedatives are required. In children the bromid of potassium is a useful remedy. It frequently controls the headache and reduces the convulsive tendency. The action of the potassium salt in producing arterial contraction is increased by the addition of chloral, which may be profitably combined with it. Applications of ice or the cold coil to the head seem at times to give some comfort and relief from pain. Antipyretics, aside from frequent gentle sponging, are rarely indicated and usually do no good.

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[1] "Chicago Medical Recorder," June, 1895.

[2] "Rif. Med." Aug. 20, 1903.

The hot bath often effectually controls the convulsions. Of late the value of drainage or mere exploration of the peritoneum in tubercular infection of that cavity has turned attention to the possible value of a similar procedure in tubercular meningitis. Some of these cases have been drained and the ventricles tapped with alleged temporary improvement. Quincke's lumbar puncture, which certainly reduces intracranial pressure and removes the fluid, has, in the cases reported by Strahan, been followed by recovery once. As a fatal ending is the only reasonable expectation, when the diagnosis has been confirmed by the lumbar puncture, the injection of iodoform emulsions or other solutions within the spinal and cranial dura may be attempted with propriety.

CHAPTER IV.

DISEASES OF THE FIRST AND SECOND CRANIAL NERVES.

DISEASES of cranial nerves are broadly divided into those which affect the *cortical centers*, those which involve the *nuclei*, and those which involve *trunks* and *peripheral portions*. The peripheral nerves extend from the nuclei to the ultimate distribution of their fibers. A part of their course lies within the mass of the brain itself. While the peripheral portion of a cranial nerve may suffer independently of its nucleus, injury to the nuclear center is always followed by degeneration in the peripheral part. It is not unusual for a peripheral nerve to be singly injured by traumatism, or even by disease, but, owing to the close anatomical and physiological relations of the cranial nuclei, the medullary centers are rarely individually diseased. Very often nuclear disease of the cranial nerves is but a portion of a more general nervous malady. The successive nuclear centers in the medulla, along the floor of the fourth ventricle, under the aqueduct of Sylvius, and on the posterior wall of the third ventricle, constitute the upward prolongation of the spinal gray matter. They may be involved with the spinal centers at the same period or at varying stages of a given general malady. The peripheral parts of the cranial nerves also participate in the general diseases which affect the spinal nerves and react similarly to infections and poisons. The analogy of the last ten cranial pairs to the spinal nerves should be clearly apprehended. The olfactory and optic nerves react more, as does the brain proper. They are, in fact, portions or lobes of the brain. To a less degree the same is true of the auditory nerve.

Diseases of the Olfactory Nerve.—The exact cortical origin of the olfactory nerve is not known. Its pathway in the brain is not clearly traced. Its disturbance, usually considered of slight importance, is often overlooked. Ordinarily, the olfactory cortical center is assigned to the uncinate gyrus, or near by, in the cornu ammonis in the floor of the lateral ventricle.[1] Certain cases of epilepsy in which an aura referable to the sense of smell was noted have presented post-mortem evidences of disease of the temporosphenoidal lobe near to or involving the uncinate convolution. The same is true of some cases of mental disease marked by hallucination of smell. It seems probable that the olfactory nerve is represented on both sides of the brain.

A loss of smell on the side opposite to a lesion in the posterior portion of the internal capsule has been noted by Féré[2] and confirmed by others. Usually, however, the lesion is on the same side of the brain, and involves the olfactory tract, the bulb, or the nervous filaments, which are distributed to the nasal vault through the cribriform plate of the ethmoid. Disease of the middle cerebral artery near its origin from the circle of Willis may cause loss of smell in the corresponding nostril. This is probably through injury to the brachia of the olfactory tract. Basilar fracture often destroys the nerve at the cribriform plate. In such cases the loss of smell is an important localizing fact. Localized meningitis, caries of bone, tumors, or abscesses may have the same effect. Hydrocephalus may seriously compress these nerves. It has been claimed that tumors situated in distant parts of the brain provoke neuritis in the olfactory tract similar to the optic neuritis usually associated with encephalic neoplasms. In old age the olfactory bulb atrophies and the sense of smell diminishes. In hysteria smell may be entirely abolished. This may be a bilateral condition and exist practically alone, but is usually unilateral and confined to the hemianesthetic side of the body. Gowers mentions some cases in which the recognition of certain odors alone was lost. Overstimulation of the sense of smell will paralyze it, and the loss may be permanent. Ordinarily, a strong stimulation for three or four minutes so blunts the sense that it no longer recognizes the particular odor, but recovers itself in about a minute. Continued exposure to strong odors usually result in permanently diminishing this special sense. Oversensitiveness is occasionally noted in neurotics, and can be cultivated. The blind and those who taste tea or inspect certain articles of commerce gain great sensitiveness in this way. Finally, the olfactory bulb may be congenitally wanting.

The **prognosis** in loss of smell from disease of the olfactory nerve is usually bad. It depends on the cause in a given case. Catarrhal nasal conditions, fifth-nerve disturbance, and hysteria must be ruled out. The use of faradism is said to have done good in a few cases. It is applied to the mucous expanse over the turbinates, and is very painful. Snuffs containing strychnin or quinin may be tried. Oversensitiveness is controlled by morphin or cocain, but their use is attended by the danger of setting up an incorrigible habit.

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[1] Zuckerkandl, "Ueber das Riechscentrum."

[2] "Arch. de Neurologie," 1885.

Diseases of the Optic Nerve.—The Visual Tract.—Only the infinitely short fibers between the layer of rods and cones and the retinal nerve-cells can properly be called peripheral optic nerves. The retro-ocular bundles that are named optic nerves by anatomists react to injury, as do other cerebral connecting tracts. Like them, if divided they never unite, while peripheral nerves unite readily under proper conditions.

The term optic nerve, with this understanding, will, however, be used as ordinarily accepted. The retina is made up of two lateral halves supplied from corresponding sides of the brain,—that is to say, the right half of each retina is in anatomical connection with the right cerebral hemisphere and the left half of each retina with the left hemisphere. At the macula lutea, or point of greatest visual activity, these halves overlap. This central part of the retina is thereby abundantly supplied from both hemispheres. As the bridge of the nose cuts off much light that would enter the pupil from that direction, the temporal or outer halves of the retinæ are rendered in part functionally inactive and their afferent fibers are less in number. The optic nerve in consequence contains many more fibers for the inner than for the outer halves of the eye-grounds.

At the chiasm in man a partial decussation takes place. The larger number of fibers, those in relation with the inner or nasal halves of the retinæ, cross to the opposite side. Those from the temporal halves of the retinæ pass backward on the same side. At this point a number of fibers enter the gray matter in the floor of the third ventricle. Back of the chiasm the temporal fibers of the right eye are accompanied by the nasal fibers of the left eye, and they together make up the right optic tract. In other words, the right optic tract contains all the fibers going to the right halves of both eyeballs. It is, then, clear that while division of an optic nerve causes absolute blindness of one eye, division of one optic tract would produce half-blindness of the corresponding sides of both retinæ. This would manifest itself in blindness in the opposite halves of the visual fields, *hemianopsia*, owing to the fact that oblique rays of light entering the pupil impinge on the opposed portion of the retina. A glance at figure 29 will make this clear. Owing to the fact that the macula has a double supply the hemianopsic field always shows an indenture at the fixing point. In such cases direct vision may not be impaired in acuity, so thoroughly is each macula supplied by both hemispheres. The dividing line between the half-fields in a hemianopsic eye is practically vertical, but may incline one way or the other to a slight extent in various individuals.

Injury dividing the chiasm longitudinally would cut off all the fibers to both nasal halves of the retinæ and produce double hemianopsia, marked by blindness for all objects to the right for the right eye and all objects to the left for the left eye. Enlargement of the pituitary gland or pressure through the floor of the third ventricle may cause this result.

In extremely rare instances a bilateral blindness in the nasal fields is caused by symmetrical lesions in the optic nerves. This condition has been usually attributed to bilateral lesion of the outer portions of the chiasm, but at this point the crossed and direct fibers are intermingled (see Fig. 29 A), and Shoemaker [1] insists that the process is due to neuritis invading the optic nerves symmetrically by way of the fibrous septa. These are quite uniform anatomical structures, as shown by Wilbrand and Sänger.

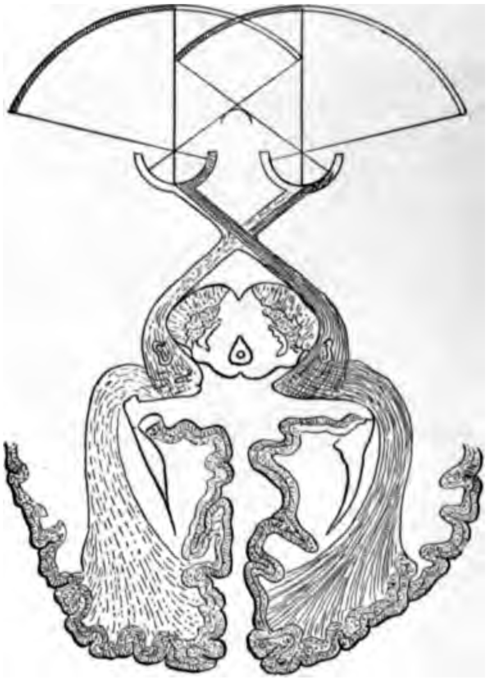
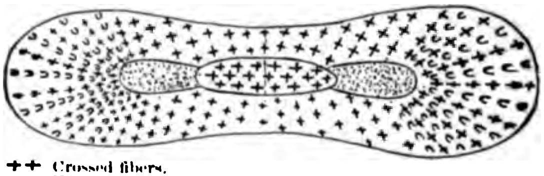


Fig. 29.—Diagram of visual paths (after Starr).



++ Crossed fibers.
⊃⊃ Uncrossed fibers.
:::: Uncrossed fibers of papillo-macular bundle.

Fig. 29 A.—Diagram of the optic chiasm, from Wilbrand and Sänger (after Henschen).

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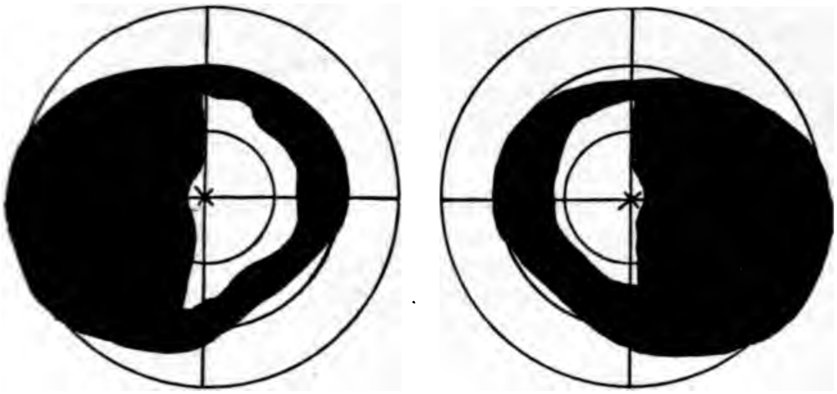
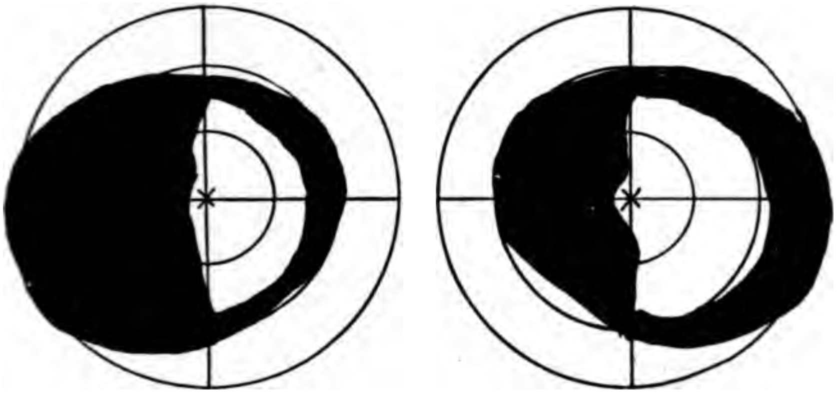
[1] "N. Y. Med. Jour.," Feb. 4, 1905.

The optic tract passing backward encircles the crus cerebri and enters the geniculate bodies, the anterior corpus quadrigeminum, and the optic thalamus of the same side. From these ganglionic bodies fibers then pass outward and backward around the posterior horn of the lateral ventricle, to end in the cortex of the cuneus, the postero-internal portion of the occipital lobe. This portion of the occipital lobe represents, therefore, half-vision for each eye and is in relation with the lateral halves of the retinæ on the same side of each eyeball. Any lesion that interrupts the visual pathway back of the chiasm, or destroys the visual centers in the cuneus, produces *lateral homonymous hemianopsia.*



Left. Right.

Fig. 30.—Blindness in both temporal fields in a case of acromegalia due to injury of the chiasm by pituitary enlargement. The nasal fields are also contracted.



Left. Right.

Fig. 31.—Homonymous lateral hemianopsia from an injury to the right occipital apex. The right lateral fields also contracted.

The cortical half-vision centers of the cuneus are in turn brought by connecting fibers into relation with higher centers for visual memories, probably situated in and about the angular gyri of the parietal lobes. In these higher visual centers both eyes are represented in each hemisphere. The parietal centers probably freely communicate through the callosal crossway. Injury to the left parietal region in right-handed persons produces loss of visual word-memories, or *word-blindness*, but does not cause hemianopsia.

The fibers which supply the macula lutea of the retina occupy at the apex of the orbit the central portion of the optic nerve in close proximity to the central artery and vein. They then become superficial on the outer side of the nerve and proceed in this position to the back of the eye. Aneurysm of the central artery or axillary inflammation of the nerve about the artery may so involve them that blindness of the center of the field develops. This condition of central blindness, or *central scotoma*, is also common in tobacco and alcoholic amblyopia. The poison seems to have the greatest effect on the most used and consequently most sensitive fibers or their related parts.

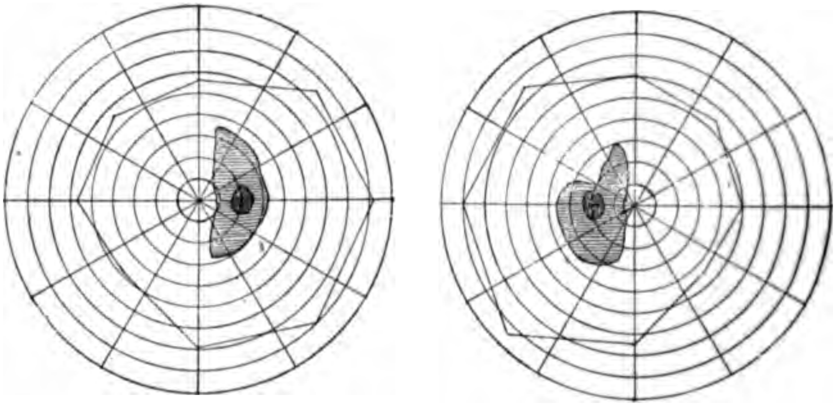

Left. Right.

Fig. 32.—Scotomata in toxic amblyopia, consisting of increased size of blind spots, which are represented by the darker shading.

The loss of vision is sometimes limited to a *quadrant* of the field. The quadrant in the field is bounded practically by lines horizontal and vertical to the fixing point, which itself is spared. A case originally presenting hemianopsia may eventually recover in part and a quadrantic loss alone remain. It is probable that these quadrants are specially represented in the occipital cortex. In organic hemianopsia usually the seeing half of the field is also more or less peripherally reduced. The blind portion of the field may not be uniformly affected, some fractional vision remaining at various points. Rarely cases have been noted in which there was hemianopsia for certain colors alone. In tobacco blindness the central scotoma, as a rule, varies for different colors.

In locating the lesion that causes hemianopsia, the *hemianopsic pupillary* *reaction* of Wernicke is of value. If the pupil responds when a narrow beam of light falls on the blind retina, the lesion is back of the geniculate bodies. The presence of this reaction indicates that the pupillary nerves are not involved. They accompany the optic tract as far as the geniculate bodies. The test must be made with great care. The reaction has a positive significance, when present, that its absence lacks. A double temporal blindness by itself is positive evidence of disease of the *chiasm*. When an *optic tract* is involved, the crus is almost invariably affected at the same time by the same lesion, so that paralytic body-symptoms on the same side as the blind field or opposite to the blind retinal half are present. Other cranial-nerve lesions are likely also to be present. Lesions of the *geniculate bodies*, the *corpora quadrigemina*, and the *optic thalami* almost invariably involve the internal capsule and produce paralysis in the body on the opposite side. A pure double hemianopsia may be produced by injury to the *cuneus*.

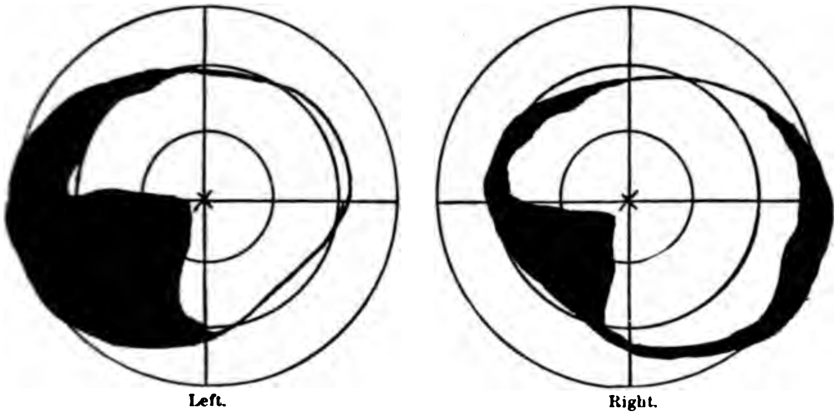


Fig. 33.—Quadrantic loss of visual fields following meningitis. Some peripheral contraction also present in the right field.

A transient hemianopsia occasionally is a symptom of migraine and lithemia. It is not impossible that hysteria may produce it, but concentric contraction of the field and changes in the color formula are usual and characteristic in hysteria. In organic hemianopsia the reductions of the field embrace form and colors equally.

The *optic nerve* is often injured by intra-orbital disease, such as tumor or aneurysm. It may be cut off at the foramen opticum by a basilar fracture, or involved by inflammation from caries of the sphenoid. An extension of inflammation down the sheath in meningitis is not rare.

The *chiasm* is injured most frequently by pituitary tumors, by syphilitic growths, and by pressure from the third ventricle in hydrocephalus. Gouty changes and interstitial hemorrhage have also been observed. Diseased carotids may press upon it.

The *optic tract* may be involved in basilar tumors or those on the internal aspect of the temporosphenoidal lobes. A patch of thickening in multiple sclerosis may affect it. The *intracerebral path* of the visual tract is often affected by tumors, hemorrhage, softening, and traumatism.

The Papilla and Retina.—Associated with or consequent upon many organic processes in the brain and spinal cord, the optic-nerve head and the retina are diseased. In two-thirds or more of the cases of encephalic tumors *papillitis*, usually double, is present at some period. It presents ophthalmoscopically the *choked disc*. The onset, often sudden, may be insidious, and in some measure the rate of development is related to the activity of the new growth. While a long-standing growth in the brain may give rise to a sudden papillitis, a chronic process in the nerve-head is never associated with acute brain disease. Of much importance is the fact that a well-marked choked disc may not be attended by much loss of vision and no symptoms may call the patient's attention to its presence. In some cases temporary loss of vision, lasting from a few minutes to several hours, has been noted. Intense optic neuritis finally affects vision in all its modes. Acuity is diminished, the field is reduced for form and color, scotomata for both are frequent, and blindness may be induced. The papilla is swollen and infiltrated with a plastic material that obscures the outline and enlarges and elevates the disc. The organization of the infiltrate, and consequent shrinking, causes the final damage to the entering nerve-fibers. Papillitis may terminate in recovery, but its legitimate end is more or less atrophy of the optic nerve with impairment of vision or complete blindness. In several cases of brain-tumor with optic neuritis, the papillitis subsided on the removal of the tumor. Even opening the skull widely may cause a reduction of the papillitis in inoperable brain tumors and prevent blindness. In other instances improvement in the brain-lesion is attended by decreasing papillitis. The intensity of the papillitis, therefore, has some diagnostic and prognostic value.

Papillitis rarely results from brain-abscess, but in basilar meningitis a *papillo-* or *neuroretinitis* is extremely frequent. In this form the choking of the disc is less marked. Orbital disease is the usual cause of unilateral papillitis, but in rare cases a one-sided optic neuritis has been caused by tumor, generally on the same side of the brain. Wilder[1] notes that in ten cases where optic neuritis was distinctly greater in one eye than in the other, the tumor was on the side of the brain corresponding to the more intense inflammation. Marcus Gunn[2] found that the tumor and unilateral papillitis were located on the same side of the head in eighteen of twenty-four cases, and with greater uniformity in the tumors that were situated anteriorly. The localizing value of bilateral papillitis is practically negative, as it is common to tumors in all parts of the brain, but is particularly frequent with cerebellar growths and those situated in the brain-axis.

Optic neuritis also occurs in toxemic conditions. It is sometimes found in anemia, often in albuminuria and in lead poisoning, and after infectious fevers. In the albuminuric form the retinal expanse is commonly invaded, but sometimes the changes are practically confined to the disc.

Regarding the causation of papillitis, many theories have been adduced and rejected. In some cases it is clearly due to irritation descending the sheath of the optic nerve from intracranial inflammation. The idea that it is due to intracranial pressure or pressure within the optic-nerve sheath in all cases has been abandoned. In toxic cases it may represent the local action of the poison. Deutschmann[1] insists that it is due to pathogenic organisms which enter from without. The question is not settled.

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[1] "Chicago Medical Recorder," June, 1894.

[2] "London Lancet," July, 1897.

Atrophy of the optic nerve may (1) follow papillitis and retinitis or choroiditis; (2) it may result from injury or inflammation to the nerve-trunk; (3) it may be associated with sclerotic disease in the brain and spinal cord; (4) it may be due to diabetes, malaria, or syphilis; and (5) it may be of unknown causation.

The atrophy consecutive to papillitis is easily understood, and of the same nature is the retinitic and choroiditic forms. Injury to the optic nerve naturally results in atrophic degeneration of the nerve-head. That form of optic atrophy found in about a tenth of the cases of locomotor ataxia, often present in paretic dementia, and not infrequent in multiple or disseminated sclerosis, has an importance quite its own. Atrophy is found in amaurotic idiocy and in the cerebellar form of family ataxia.

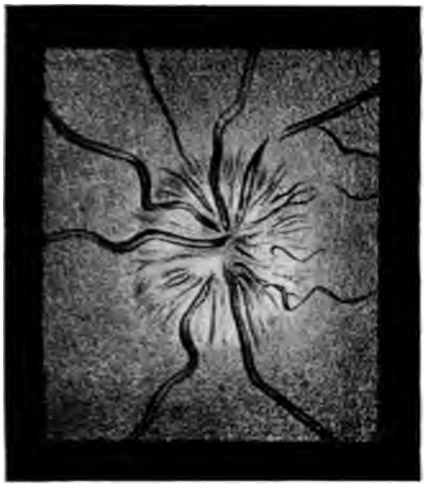

Fig. 34.—Optic neuritis.

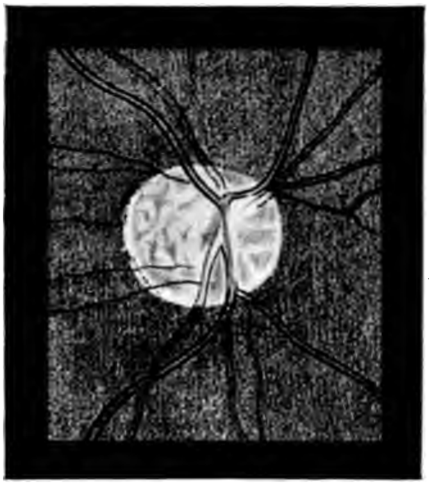

Fig. 35.—Atrophy of the optic nerve.

The symptoms and ophthalmoscopic pictures are tolerably uniform for the various forms. In the variety associated with tabes the disc is often grayish, translucent, and shows the stipling of the lamina cribrosa. In the postpapillitic form the translucency and stipling are less marked. In amaurotic idiocy there is a peculiar bluish spot at the site of the macula about twice the size of the disc, presenting in its center a brownish-red spot strongly contrasting with its surrounding patch and resembling a central embolism or hemorrhage. At the same time the disc is atrophic. In all forms of atrophy the disc is sharply outlined from the surrounding rètina by its pallor and the vessels are diminished in size.

Functional and Toxic Blindness.—The peculiarities of hysterical blindness will be detailed in the description of that disease. In such cases the loss of vision may come on slowly or suddenly. It is usually unilateral and most marked on the paralytic or anesthetic side of the body, but the opposite eye is nearly always somewhat affected. The characteristics are contracted fields and inversion of the color formula. When apparently absolute, by using prisms or other suitable means, it can be demonstrated that the eye operates properly, but that ocular impressions are ordinarily neglected by the higher visual centers, where the disturbance must be located.

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[1] "Ueber Neuritis Optica," 1887.

Disease in branches of the *fifth nerve* is sometimes attended by partial blindness. This is marked by considerable concentric contraction of the field and some loss of acuity of vision. It is principally associated with disturbance in the dental branches, particularly those to the molar teeth.

Acute *anemia*, the result of excessive hemorrhage, sometimes induces blindness that may be complete and permanent.

A number of general *toxic states*, such as uremia and syphilis, and those due to quinin and lead, may cause amblyopia. Uremic blindness is often of sudden onset, but may be preceded by dimness or haziness of vision. The fundus in such cases usually presents albuminuric retinitis and Bright's disease is present. Temporary or recurrent blindness in syphilitic involvement of the brain is sometimes a valuable early diagnostic symptom of that condition and also of brain-tumor. Early correction of the toxic state in the various poisonings usually promptly results in a return of vision, but the persistence of toxemia may induce permanent blindness.

Excepting in the albuminuric cases, the fundus at first ordinarily presents nothing abnormal. Sometimes pupillary symptoms are absent; sometimes there is dilatation and immobility. The difference seems to be due to the essential involvement of the cortical cells in the first case, and of the retinal cells, or of both, in the second.

No attempt is made in the foregoing to exhaustively treat of the diseases of the visual tract and retina. The involvement of the second cranial nerve is commonly only a part of a neurological case, but has a symptomatic value of great importance. From this standpoint the various features that relate to diseases of the nervous system have been grouped.

CHAPTER V.

DISEASES OF THE OCULAR NERVES—THE THIRD, FOURTH, AND SIXTH CRANIAL PAIRS.

Anatomical Considerations.—The muscles of the eye are supplied by the third, fourth, and sixth cranial pairs and the sympathetic. The third, fourth, and sixth nerves arise from collections of cells ranged from before backward, under the posterior portion of the middle ventricle, the aqueduct of Sylvius, and the anterior angle of the fourth ventricle.

They have in common the function of controlling the movements of the eyeballs. In addition, the third nerve governs contraction of the iris and the ciliary muscle, and elevation of the upper eyelid. The sympathetic, through the optic ganglion, dilates the pupil. Starting from before backward in the nuclei of the *third nerve*, we find represented (1) *ciliary* and then (2) *pupillary* contractions. The centers for the extrinsic ocular muscles follow presumably in the following order: The internal rectus, the superior rectus, the elevator of the eyelid, the inferior rectus, and the inferior oblique. The *fourth nerve* is distributed solely to the superior oblique, and its center is placed close behind those for the third nerve. At a little distance caudad under the floor of the fourth ventricle is the center for the *sixth nerve*, which controls the external rectus. All these centers are in close functional and anatomical relationship, and can reasonably be considered as a single mechanism, made up of three portions. The first is the iridociliary, the second embraces the centers for the muscles of convergence, and the third comprises the nucleus for the sixth nerve, which controls the external rectus, the only divergent ocular muscle. Ocular movements are also represented in the frontal motor-cortex. The nuclear centers are connected with the higher levels through the motor radiations and the internal capsule. The muscles governed by the third nerve are also brought into relation with the orbicular muscles of the lids by nuclear connection with the facial nerve. The accommodation and light reflex are thereby correlated to the act of winking and the position of the palpebral curtains.

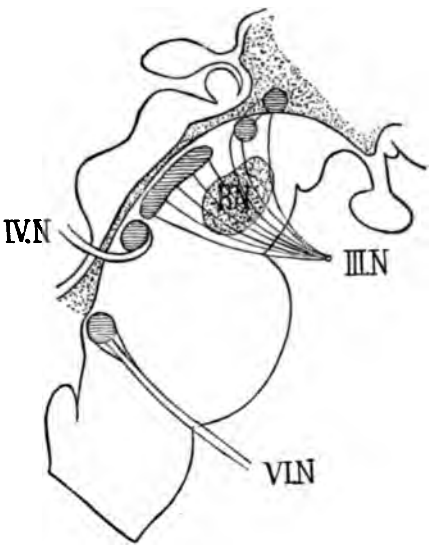

Fig. 36.—Diagrammatic longitudinal section of the mid-brain, showing the relation of the nuclear centers for the ocular muscles (after Brissaud).

Based upon the studies of Schaefer, Unverricht, Danillo, Munk, and his own experiments, Roux [1] asserts that the oculomotor apparatus has a double cortical representation; first an anterior one at the foot of the second frontal convolution, and, second, a posterior center in the occipital region. Von Bechterew [2] also contends that irritation of the anterior margin of the occipital lobe in the dog produces narrowing of the pupil and increase of accommodative effort.

The various ocular muscles serve to move the globe in the orbit in the directions indicated by their names; but the superior and inferior recti, owing to the oblique direction from the apex of the orbit to their insertion, also draw the eyeball toward the nose and rotate it inward. This is counteracted by the oblique muscles and the external rectus, but the oblique muscles also act in convergence. Convergence of the eyes, necessary for all close vision, is thus much better provided for than the opposite action. Voluntary divergent squint is impossible.

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[1] "Arch. de Neurol.," Sept., 1899.

[2] "Neurolog. Centralblatt," May, 1900.

The motor nerves of the eye may be diseased (1) at their nuclear origin, (2) in their intracerebral course, (3) in their intracranial course from apparent origin to their cranial outlets, and (4) within the orbit. Four groups of symptoms are produced: (1) Pupillary variations, (2) disturbance of accommodation, (3) muscular incompetence and squint, (4) double vision. It is by the study of these symptoms, their mutual combinations, and the association or absence of other cerebral indications that a distinctive and localizing diagnosis is possible. We will first consider the individual nerves.

Ocular Palsies.—Complete division of the *third nerve* paralyzes all the extrinsic muscles but the external rectus and the superior oblique. The eye, in consequence, can be turned strongly to the outer canthus, and eventually becomes fixed in that position. The eyelid droops in complete ptosis, which the patient tries to remedy by the action of the frontalis. The pupil is widely dilated by the unopposed action of the sympathetic, and does not react for light or accommodation. The eye, unless strongly myopic, is incapable of near vision through the loss of accommodation due to paralysis of the ciliary muscle. There is double vision excepting when the sound eye is made to correspond with the direction of the one paralyzed.

Division of the *fourth nerve* allows the eyeball at the top to slightly rotate outward on the anteroposterior axis, which corresponds practically to the line of direct forward vision. When fixed in this position by the action of the inferior oblique and the inferior rectus, which act together, the rotation can sometimes be observed if carefully looked for, but the index to this paralysis is in the diplopia, to be studied later. In attempts at downward convergent vision the inward rotation of the eyeball fails to take place.

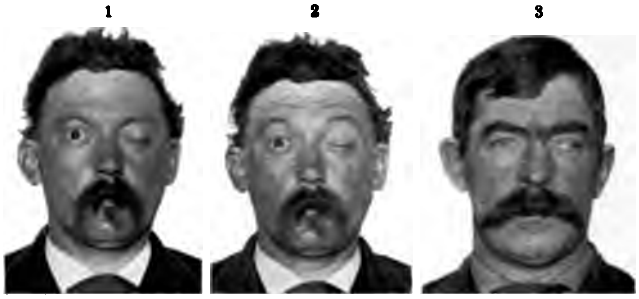

Fig. 37.—Paralysis of the left third cranial nerve. 1, Ptosis; 2, ineffectual attempt of frontalis to overcome ptosis; 3 shows partial ptosis and outward deviation of affected eyeball.

When the *sixth nerve* is divided, the eye can not be turned outward from a line marking direct forward vision, and later the eye becomes fixed in a strong inward squint that may carry the pupil to the internal canthus. The diplopia is marked.

Destructive injury to the *sympathetic* produces great narrowing of the pupil, as the pupillary sphincter, innervated by the third nerve, escapes. At the same time the eyeball becomes prominent or slightly exophthalmic by reason of the paralysis of Müller's muscles and the retro-ocular turgescence. The pupil does not react for light or for pain.

Diagnosis of Ocular Palsies.—When the lesion is complete and has existed some time, difficulty in deciding which extrinsic muscle is at fault is not very great. In partial lesions and when the condition is vacillating or slight in degree, a careful examination is required. We have two important indications—namely, the *habitual position of the head* and the *diplopia.* When an ocular muscle is weakened, the patient unconsciously and constantly so carries the head that the least possible work is required of the paretic muscle. For instance, if the right external rectus is involved, the patient will turn the head to the right, thereby relaxing the injured muscle. The rule as laid down by Landholt [1] is that *the direction of the head corresponds in every way to the physiological action of the paralyzed muscle.*

If the extended lines of the visual axes for both eyes do not attain the same fixed point, *diplopia* results. The sound eye sees the fixed object clearly and the mind refers the image to the proper position in space. The divergent or convergent eye sees the fixed object indistinctly, as the image does not fall on the sensitive spot of the retina and the mind projects it to a wrong position in space,—namely, to the position by experience associated with the particular part of the retina in operation. In diplopia of long standing the mind learns to neglect the weaker, false image, and the patient may be unaware of his double vision. The eye also usually becomes more and more divergent, presenting the comparatively insensitive retinal periphery to the fixed object, and this assists in the mental neglect of its image. When the muscle weakness is very slight, diplopia only occurs on quick movements of the eyes or in extreme range of the eye in the direction of the affected muscle's action, and then it may be but momentary, the muscle under the stimulus of attention gradually drawing the eye into line. This is often accomplished in a jerky manner. The eyeball presents a number of oscillations which may terminate in the proper position, or the globe may swing back to the abnormal position, the muscle being quite exhausted. The double vision and the faulty projection at first invariably give rise to pronounced ocular vertigo.

In examining a case of diplopia the first question is whether it is monocular. Closing one eye prevents double vision unless it is confined to the open eye, in which case, if not due to defective media or faulty curvatures, it is almost surely hysterical. Place the patient in a good light, have him hold the head motionless, and let him follow with both eyes the point of your finger through a circular range about a foot from his face. At some angle the two eyes will fail to maintain parallelism, or jerky movements of one eye may be noticed. The point is to determine which is the affected eye. Cut off the patient's line of sight from one eye with a card held at such a distance from it that its motion can still be observed, and have the patient fix both eyes upon your finger in the direction toward which conjugate movements failed. If the sound eye be covered, it will overact and be turned farther to the side than is required. This overaction, or *secondary deviation*, is due to the fact that the stimulation necessary to draw the affected eye in the direction of the weak muscle is more than sufficient for the corresponding sound muscle. If you have covered the weak eye in the same way it will not move outward far enough when the finger is fixed by the sound eye, and for a similar reason. The direction of failure in the affected eye, therefore, points to the weakened muscle. In marked squint of this character the affected eye often presents a much wider range of motion when the sound eye is covered than when binocular vision is attempted. Apparently the volitional attempt is stronger when the muscle is not disturbed by two images. Overaction of the sound eye, however, occurs, as in the card-test.

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[1] "Brit. Med. Jour.," Sept. 15, 1894.

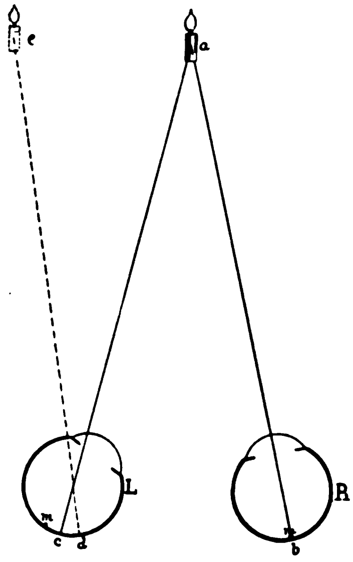

Fig. 38.

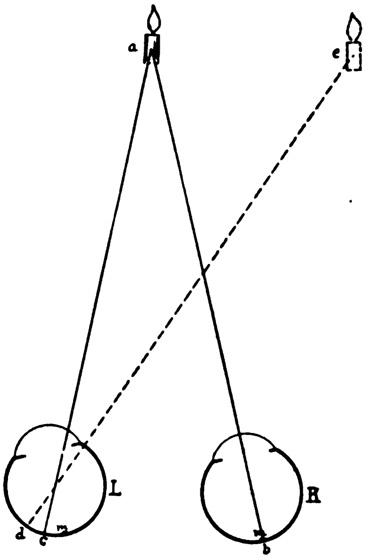

Fig. 39.

Fig. 38.—Convergent squint. The ray of light, a c, falls to the right of the sensitive spot, m, of the inturned left eye, L. Such stimulus has always been recognized by the mind as emanating from objects to the left, and not realizing the disordered position of the ocular globe a false image, e, is projected an additional distance to the left, measuring from c as the operating sensitive spot, in the line d e.

Fig. 39.—Divergent squint. The ray of light, a c, falls to the left of the macula, m, of the outwardly turned left eye, L. Such stimulus has always been associated with objects to the right, and the false image, e, is projected to the right, c, acting on the sensitive spot at the time.

The *diplopia test* is made as follows: Place the patient facing a candle or bright object, such as a narrow strip of white paper the size of a lead-pencil, placed on a dark background at a distance of, say, twenty feet. Cover one eye with a red glass. If there is double vision, the red image corresponds to the covered eye. If the red image and the covered eye are on the same side, the diplopia is *simple*; if on opposite sides, the diplopia is *crossed*. When the eyes converge, the diplopia is simple; when the eyes diverge, the diplopia is crossed. Gowers' rule is: "*When the visual lines, the prolonged visual axes, cross, the diplopia is not* *crossed.*" If, now, the fixing object remaining stationary, the head be turned one way and another,—in some given direction the images will separate and in the opposite direction approach and perhaps fuse. Landholt [1] lays down this rule: "*The affected eye is that in the direction of whose image the diplopia increases.*" For instance, in simple diplopia, the right image corresponds to the right eye. If the diplopia increases on looking to the right, the right eye is affected. In crossed diplopia the left image corresponds to the right eye. If the images separate on looking to the left, the right eye is at fault because its image corresponds to the direction of the increased diplopia. The same thing is true in vertical diplopia.

He also lays down this rule: "*The paralyzed muscle is the one which would give to the eye the direction of the false image.*" The false image, of course, belongs to the affected eye. For instance, in simple diplopia with the right eye affected, it is the external rectus that is at fault, as it is this muscle which would, if competent, direct the eye to the position of the false image at the right of the true image. Involvement of the oblique is similarly shown. If the patient indicates that the image of the right eye, in simple diplopia, is to the right and its upper end leans to the left, we know, by the application of this rule, that the right superior oblique is deficient. An opposite condition would point to the inferior oblique and inferior rectus, which operate together.

The *location of the lesion* determines the distribution of symptoms and through them is clinically deciphered. A lesion involving the *nuclei* under the aqueduct of Sylvius is almost invariably attended by bilateral symmetrical eye conditions. This is due to the anatomical proximity of the centers on each side of the middle line and to their physiological relations. A lesion which destroys accommodation and pupillary contractions affects the *most forward group* of nuclei. If the *middle group* is involved, all the extrinsic muscles of the eyes are disabled, even the external recti, as this group has a controlling influence over the nucleus of the sixth nerve. If the lesion fall upon the *sixth nucleus* alone, double convergent squint is produced. In the same way, if some one or several of the conjugate movements of the eyes be lost, or a double ptosis is present, the lesion is nuclear unless movements of the head and trunk are lost at the same time. In such case the lesion is probably above the nucleus in the internal capsule or in the cortex.

The three ocular nerves leave their grouped centers by widely diverging *routes*, and may be cut off separately and unilaterally by encephalic conditions, such as hemorrhage, inflammation, or new growths. Such lesions invariably implicate other structures, and give rise to numerous symptoms other than those referable to the ocular nerve. For instance, a lesion in the peduncle in the region of the red nucleus would disturb the motor tracts in the crus and produce paralysis on the opposite side of the body, with paralysis of the third nerve, ptosis, etc., on the same side as the lesion. This, by Charcot, is denominated the symptom group of Weber (fig. 36).

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[1] *Loc. cit.*

In basilar inflammations and injuries, and particularly in syphilitic meningitis, the ocular nerves are likely to be injured. The sixth, from its long and exposed course, is especially prone to injury and is the most frequently diseased nerve of this group. Lesions in this position are usually unilateral, and if bilateral differ, as a rule, in degree on the two sides. Here injury to the third nerve necessarily involves all its branches and functions. A lesion which could select from the nerve-trunk the pupillary fibers, for instance, is inconceivable. Diffuse cerebral symptoms, such as headache, vertigo, nausea, stupor, convulsions, and optic neuritis, are also commonly present. At the *apex of the orbit* all the ocular nerves, with the ophthalmic division of the fifth and the optic nerve, are closely approximated. Injury or disease at this point would, therefore, determine complete internal and external ophthalmoplegia, impair vision, and give rise to sensory disturbances in the distribution of the supra-orbital nerve. Pressure at the same time on the return orbital circulation congests the retinal veins and produces some exophthalmos. After the nerves have separated *in the orbit*, any one or more of them may be injured by traumatism or local disease. Their branches may be singly selected. The diagnosis depends upon the muscles involved, and the anatomical relations of the nerves and their branches within the orbit.

Causes of Ocular Palsies.—The conditions which give rise to palsies of the ocular muscles are very numerous. *In the orbit* traumatism and new growths are frequently causal of the loss of ocular movements. A blow over the eye is sometimes followed by paralysis of the levator palpebræ superioris. Exposures to cold and the rheumatic states are sometimes followed by an ophthalmoplegia, which is probably due to a peripheral neuritis. This form may be present in multiple neuritis from alcohol, and probably from other poisons, and may involve few or many of the muscles. Orbital growths usually are marked by exophthalmos. Cervical, maxillary, and cranial tumors may invade the orbit and there produce these local symptoms.

In their basilar course the motor nerves of the eye are frequently injured by meningitis, and especially by syphilitic disease. Basilar fracture, carotid aneurysm, or the pressure of neighboring intracranial growths may also implicate them in this locality.

At the nuclei multiple sclerosis, bulbar palsy, polioencephalitis superior, and locomotor ataxia frequently cause ocular palsies. A temporary ptosis or diplopia is often an early symptom of tabes, and should always arouse suspicion of it or of syphilis. The exact mechanism and location of the tabetic disturbance which produces the Argyll-Robertson phenomenon is not understood, but presumably the anterior cell-group, controlling pupillary and ciliary contractions, is involved.

A peculiar *periodically recurring* or *relapsing palsy* of the third nerve is sometimes encountered. It is more frequent in females, involves only one eye, lasts from one to six months, and returns at regular intervals, often during the entire life of the patient. Its pathology is not known, but the nerve-trunk has several times been found to be diseased. Traces of this palsy, in some cases, can be detected during the intervals of practical freedom from the trouble. It has been attributed to hysteria in certain instances, and is frequently associated with migraine, the so-called *ophthalmoplegic migraine.*

Lesions of the cortex or of the internal capsule producing hemiplegia are often attended by conjugate deviation of the head and eyes toward the sound side of the body, but if the paralyzed side present convulsions due to cerebral irritation, the eyes are then spasmodically drawn to that side. A lesion lower down in the pontine region would be marked by a crossed deviation, the eyes looking to the paralyzed side of the body, and in convulsions turning to the sound side of the body.

Ocular Muscle Spasms.—The ocular muscles are usually associated with the other muscles in generalized convulsions, the eyes turning to the side most vigorously affected. Individual muscles alone are affected in very rare instances. The levator or a rectus may thus be spasmodically involved for years. Sometimes the ocular muscles participate in facial tics and spasmodic torticollis. In hysteria the eyes are frequently rolled upward and outward, or strongly converged during convulsive manifestations of the disease, and these ocular positions may remain for long periods of time after the convulsion has subsided. A more common eye condition in hysteria is one of spurious double ptosis. It is really a spasm of the orbiculares. The patient apparently tries to overcome it by raising the brows with the frontalis. An attempt to raise the lids with the finger at once develops resistance, which in paralytic ptosis is never present.

Nystagmus consists in rapid rhythmical involuntary oscillations of both eyeballs, due to spasmodic action of the ocular muscles. When both eyes are affected, the movements are similar and synchronous. The spasmodic movements are commonly horizontal, less frequently rotary, and only rarely vertical. When the eye vibrates in one plane, it ordinarily moves outward quickly and more slowly returns. The oscillations vary in number from fifty to two hundred or more a minute, and in extent from one to ten millimeters. In uremic coma the eyes will sometimes be noticed in a rhythmical movement which carries them through their widest lateral range about ten times a minute. Nystagmus may be constant or only provoked by calling forth a particular movement of the eyes, as by directing them upward or outward. This latter must not be confused with the jerky, unrhythmical, and momentary movements of weakened muscles.

The causes and significance of nystagmus are often obscure. It may be acquired as the sequel of any condition which greatly impairs vision. Corneal and lenticular opacities, choroiditis, and retinitis may precede it, especially if occurring in childhood. Albinos frequently present nystagmus. Miners who work in cramped positions with poor illumination acquire it, probably as a fatigue neurosis through the overstrain of certain eye-muscles in attempting to keep the work in view. In them it is often only present when the particular attitude which their work requires is assumed.

Nystagmus is present incidentally or regularly in a long list of nervous diseases which have neither location nor pathological anatomy in common. It is almost a cardinal symptom in insular sclerosis and in hereditary ataxia of the Friedreich type. It is common in cerebellar tumor and tumors involving the corpora quadrigemina and optic thalami. Occasionally it attends sinus thrombosis, meningitis, meningeal hemorrhage, and variously seated cerebral hemorrhages, softenings, and tumors. Hysteria has also furnished rare cases of persistent nystagmic ocular spasm.

The treatment of ocular palsies depends upon the causal condition and is often most unsatisfactory. New growths and traumata are surgical conditions for the most part. Syphilitic palsies may readily yield to treatment, whether due to basilar or central involvement, but only too often recur, yield again, and finally become permanent. The neuritic form is treated as a part of the general intoxication usually at the bottom of the disease. A persisting ocular deviation is often benefited by a tenotomy. Muscle cutting should always be preceded by exercise of the muscles with prisms, which sometimes is helpful, and by general treatment. Nystagmus, excepting in the unusual cases where it is due to meningeal and sinus diseases, or other curable lesion, is practically unyielding to all measures. Miners' nystagmus usually ceases when the occupation is changed.

CHAPTER VI.

DISEASES OF THE TRIFACIAL NERVE.

Anatomical Considerations.—The fifth nerves represent the sensory portions of all the motor cranial nerves. Their sensory distribution embraces most of the skin of the head and face, all their mucous membrane-lined cavities, and the cerebral meninges in part. The exact limits of this sensory field have been worked out by Cushing in a number of cases subjected to extirpation of the ganglion of Gasser, and are shown in Fig. 39 A. In addition, through at least the chorda tympani, the fifth subserves the special sense of taste. Its small motor root innervates the mandibular muscles of mastication. The nuclear origin of the fifth nerve in the medulla is correspondingly extensive. The smaller motor nucleus is situated under the floor of the fourth ventricle near its lateral angle, with an upward extension as high as the corpora quadrigemina. Outside of this is the larger sensory nucleus, which is connected continuously with nuclear gray matter as low as the fourth cervical spinal segment. These centra are brought into relation with the cerebellum and with the cerebral cortex by upward radiations. For the motor portion the cortical centers are at the foot of the anterior central convolution. The sensory cortical repre-

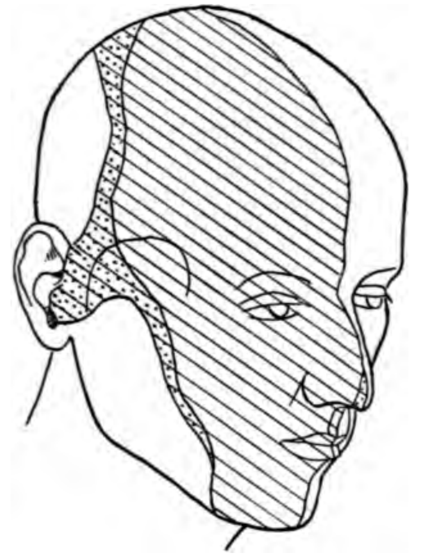

Fig. 39 A.—Diagram showing the normal (average) field of postoperative cutaneous anesthesia. The shaded area, including tragus and anterior wall of meatus, remains anesthetic to tactual (hair esthesiometer) stimuli. The dotted strip gives the impression of touch or pressure to pain stimuli (needle), with few if any actual pain points (Cushing, in Bull. Johns Hopkins Hosp., July-Aug., 1904).

sentation is not clearly known. The sensory and motor trunks leave the surface of the pons separately, though side by side, and only after the sensory portion has passed through the Gasserian ganglion does the motor trunk join the third trigeminal division.

According to Gowers and others, the strictly gustatory portion follows a most extraordinary course before, as the chorda tympani, it reaches the lingual nerve and is distributed to the anterior portion of the tongue. At first contained in the sensory trunk, it apparently passes from the Gasserian enlargement with the middle or superior maxillary branch. Thence it drops into the sphenopalatine or Meckel's ganglion, and turns backward in the form of the Vidian nerve, to penetrate the petrous portion of the temporal bone and join the facial nerve in the Fallopian aqueduct. It follows the facial nearly to its exit at the stylomastoid foramen, where it turns sharply upward, reaching the tympanic cavity, which it leaves by the Gasserian fissure. It then descends between the pterygoid muscles and joins the lingual branch of the inferior maxillary or third trifacial division, to be distributed to the anterior portion of the tongue. The base of the tongue and faucial pillars also recognize taste impressions. It is supposed that these sensations reach the trunk of the fifth by way of the glossopharyngeal nerve through connections with Meckel's ganglion. We know certainly that division of the sensory trunk of the fifth abolishes all taste sensation on the corresponding side of the tongue at least temporarily, but Cushing,[1] after an elaborate study of twenty-six cases of extirpation of Gasser's ganglion, believes that the sense of taste ultimately returns to the tongue, and that the gustatory path is not by way of the fifth nerve.

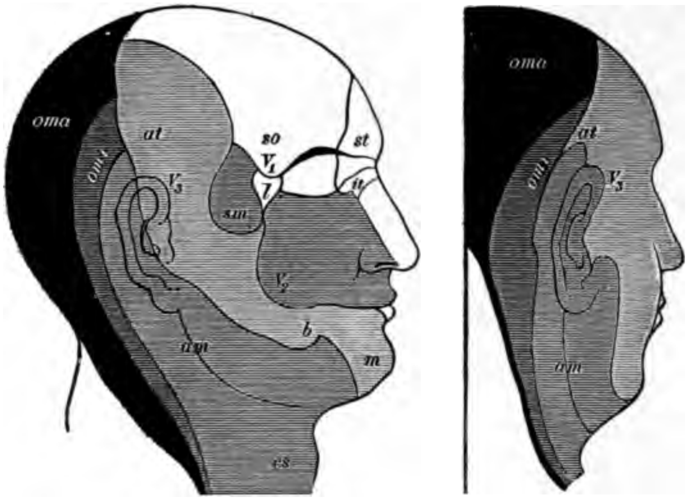

Fig. 40.—Distribution of the sensory cutaneous nerves on the head: V_1, V_2, V_3, The three branches of the trigeminus; *at*, auriculotemporal; *so*, supraorbital; *st*, supratrochlear; *it*, infratrochlear; *l*, lacrymal; *m*, mental; *b*, buccinator; *am*, auricularis magnus; *sm*, subcutaneous malæ; *oma*, occipitalis major; *omi*, occipitalis minor; *cs*, superficial cervical (after Hirt).

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[1] "Johns Hopkins Hosp. Bull.," July-Aug., 1904.

Cortical Diseases of the Fifth Nerve.—Lesions of the portion of the motor cortex associated with the fifth nerve produce spasm or paralysis according as the lesion is irritative or destructive. The involved muscles are the pterygoids, the temporal, and the masseter. In generalized convulsions they are ordinarily involved. Very rarely they are the sole seat of motor disturbance presenting a *masticatory spasm or paralysis*. The spasm may be tonic, as in trismus, or repeated at varying intervals. The teeth are tightly clenched, and the masseters and temporals stand out firm and hard. In the clonic variety, as in the chattering of a chill, the lower jaw is sharply moved laterally or vertically. Vigorous grinding of the teeth may be present. This is sometimes seen in the late stages of paretic dementia and in other wide-spread organic brain disease. Single or repeated spasms may have their origin in local disease of the jaws, such as periosteitis or toothache, and are then reflexly produced.

Masticatory paralysis of cortical origin is extremely rare. The cortical lesions have almost invariably been found to be bilateral, but Hirt[1] has reported a case of complete mandibular palsy due to a left-sided lesion at the foot of the ascending frontal convolution.

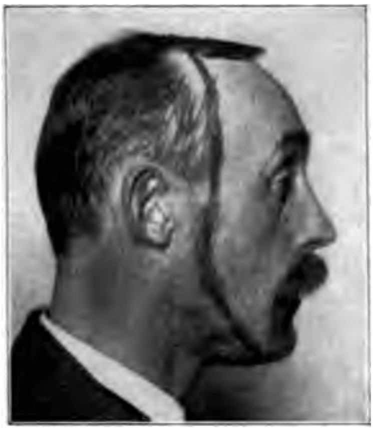

Fig. 41.—Case of nuclear disease of the fifth cranial nerve in a case of tabes, showing area of cutaneous anesthesia; some facial atrophy is also present.

Nuclear disease of the fifth nerve is usually a part of a group of bulbar symptoms. Masticatory paralysis arising from this source is extremely rare. Progressive bulbar palsy and pontine tumors may cause it, but other cranial nerves are almost invariably affected at the same time. Multiple sclerosis and tabes may and often do affect the fifth nucleus, causing bilateral sensory and motor symptoms in the area of its distribution.

Peripheral intracranial affections of the trigeminus may involve the stem, the Gasserian ganglion, or the three branches of the nerve at their exits from the skull. The differential diagnosis as to location may be impossible if adjoining nerves or structures are uninjured or present no indications. A description of the symptoms arising from injury to the trunk will therefore apply to disease of the Gasserian ganglion or of the three branches at their cranial exits. Growths and inflammatory processes are the usual causes of this form of trifacial disease.

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[1] "Berlin. klin. Wochens.," 1887.

The motor symptoms are those of paresis or paralysis of the mandibular muscles. The jaws can not be closed or can not be firmly held together when closed. The combined strength of the jaw-muscles is immense, and considerable impairment may go unnoticed. In complete bilateral paralysis the jaw droops, but can at first be raised by the action of the facial muscles, especially the buccinators and orbicularis oris. If the palsy is one-sided, the jaw may still be raised by the unilateral action of the uninjured side, but the bite is feeble. Attempts to bring the paralyzed pterygoids into play fail to produce grinding movements, so that the jaw can not be forcibly advanced from the impaired side or thrust in the opposite direction. In long-standing cases contracture in the muscles which depress the jaw may permanently hold the mouth open.

Interference with sensation may be partial or complete. In the latter case all parts of the face, head, nasal fossæ, conjunctivæ, mouth, and tongue supplied by the fifth nerve are insensitive, and taste is abolished on that side of the tongue and oral cavity. The sneezing reflex is abolished on the anesthetic side, as well as the gagging reflex caused by irritating the soft palate. Instruments may be passed into and through the nasal chambers without eliciting sensation, and ammonia fumes, etc., no longer cause irritation. Prodromal pricking, tingling, and burning usually precede the anesthesia. Frequently, when the loss of sensation is pronounced, so that the patient no longer feels a pin-prick, complaints are made of pain and burning in the anesthetic area,—anæsthesia dolorosa. In one case observed by the writer, while general sensation was abolished in all its modes and tenses, muscular sense remained. A touch or prick was not perceived, but the slightest motion communicated to any facial muscle was instantly recognized, apparently through the uninjured seventh nerve. *Trophic disturbances* are the rule, but, ordinarily, they are slight in degree. The insensitive conjunctiva and cornea are easily irritated and prone to ulceration that may reach a destructive grade. The nasal and lacrimal secretions are defective and the mucous membrane dry. In the nose this dryness impairs the sense of smell. The paralyzed side of the tongue is thickly furred, due partly, but not wholly, to the fact that food is only chewed on the sound side. The salivary secretion may be greatly diminished. Herpetic eruptions in the cutaneous distribution are frequent, and when the ganglion or branches are diseased and the conjunctiva is involved, constitute a serious feature, as ophthalmia and complete loss of the eye may ensue.

Facial hemiatrophy may follow injury and disease of the fifth nerve. In this rare deformity the wasting is always limited to the distribution of the trifacial, and is usually most intense in the field of the middle and inferior divisions. The alleged causes in numerous instances, such as blows on the head and face, infectious fevers, exposure to cold, facial erysipelas, osteitis of the jaws, etc., are capable of seriously influencing the nerve. In several cases histological changes in the trunk, ganglion, or branches have been demonstrated, and atrophy has followed division of the root of the trigeminus in man and animals. It may begin in a widening sclerodermic patch on the side of the face, but more commonly the entire half of the face gradually diminishes. The loss affects both dermal and osseous structures and less markedly the muscles, which may escape entirely. The opposite side of the face may finally become involved, though this is rare. The disease develops usually before adult life, but may appear at any age. It produces a most notable difference of appearance on the two sides. The atrophic half lacks the proportions of the sound side in every particular. The condition is usually most marked in the lower portion of the face, gradually lessening upward so that the brow may show almost no discrepancy on the two sides. The skin is thinned notably, sometimes to a half or quarter of its proper thickness; the muscles are sometimes reduced in size and strength; the lower jaw may be a third smaller on the affected side. The teeth are often lost. As the skin is closely applied to the muscles and the bony conformation, a cadaverous appearance is presented that may be strikingly at variance with the plump, healthy side, and is sometimes sharply marked by a furrow at the middle line of the brow and chin. The nose, chin, and mouth deviate to the affected side. While the orbital and palpebral structures are frequently wasted, the eyeball is affected only in rare cases, but has been observed wasted and even destroyed. The disease is progressive for years, but may come to a standstill at any time, and again advance. It seems to be unmodified by treatment, but the continuous use of thyroids may be tried with some prospect of retarding the progress of the wasting, especially in those cases showing scleroderma. Paraffin has been injected in a number of instances to restore the contours of the face purely for cosmetic effect.

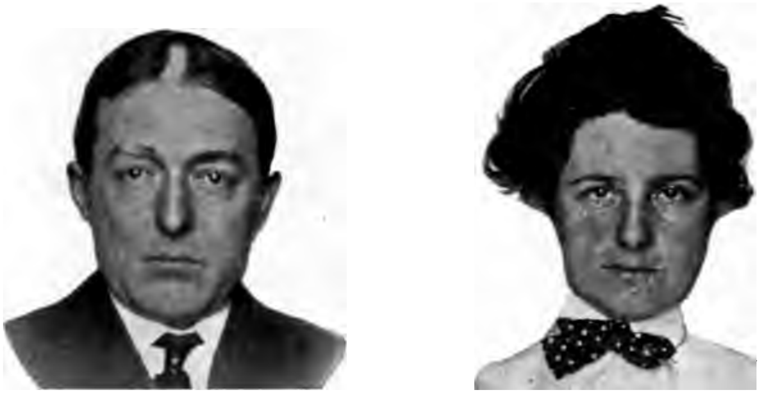

Fig. 42.—Two early cases of facial hemiatrophy.

Disease of the trifacial branches is extremely common. Their course through bony channels, serving to protect them admirably under ordinary conditions, exposes them to pressure from inflammatory states, to injury by concussing blows, and to laceration from fractures involving the cranial and facial bones. Their proximity to the nasal, buccal, and pharyngeal cavities, always containing the potential factors of infection, is a local disadvantage. Finally, they are distributed to the most exposed portion of the cutaneous expanse, where, thinly covered, they rest upon unyielding structures.

They are very often the seat of *neuralgic pain*, which will be more particularly considered in the section on Symptomatic Disorders of the Nervous System, Part VIII. It is probable, however, that a nerve, the seat of long-continued neuralgic pain, symptomatic, perhaps, of a general blood state,—as malaria, for instance,—may eventually become histologically changed and organically diseased. A *neuritis* may be thus established which is usually marked by sharply-defined anatomical areas of hypersensitiveness or anesthesia. Dystrophic changes in the dermal structures, such as scaliness, herpes, and falling or discoloration of the hair of the eyebrow and beard, are frequently encountered. The glands supplied by the given nerve over- or underact as the condition in the nerve is irritative or destructive. Neuritis may also be set up by extension from a neighboring inflammation in the orbit, antrum, or jaws. The dental branches are particularly liable to infection, injury, and irritation, which in the case of molar teeth is not infrequently the cause of pain referred to other branches of the trifacial than the one immediately concerned.

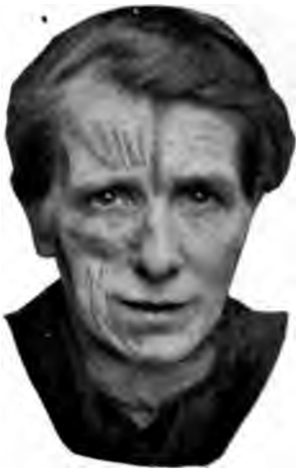

Fig. 43.—Case of facial hemiatrophy (after Yonge).

Injury to the lingual branch of the third division of the fifth nerve, if it occurs below the junction of the chorda tympani, produces loss of taste on the anterior portion of the tongue on the same side, in addition to the loss of general sensation and trophic disturbance in its anatomical area of distribution.

The *motor fibers* of the trifacial, being entirely confined to the third division, suffer with it. Paresis or paralysis of the muscles of mastication on the same side follows. The prominence and hardness of the masseter and temporal do not take place when the patient attempts to clench the teeth, and deviation of the chin to the opposite side can be but feebly produced or is lacking. Destruction of the *motor root*, either above or below the ganglion or at the bulbar nucleus, gives rise to muscular atrophy in the muscles of mastication, and to the reaction of degeneration upon their electrical stimulation. A cortical lesion causes no such degeneration.

CHAPTER VII.

DISEASES OF THE FACIAL NERVE.

Anatomical Considerations.—The seventh cranial nerve has its cortical origin or representation in the lower Rolandic region. The nuclear center is situated under the floor of the fourth ventricle, to the inner side of the ascending root of the fifth nerve. The pathways between nuclei and cortex decussate in the median raphe (Edinger). From the nuclear cells the nerve passes close to the nucleus of the sixth nerve, and, descending thence through the pons, emerges in the furrow between the pons and medulla outside the sixth nerve, closely accompanied by the eighth or auditory nerve, with which it proceeds directly to the internal auditory meatus. Its relation to the sixth nucleus and its proximity to the sixth nerve on emergence explain the frequent association of these nerves in disease. The parallel course of the auditory and facial from medulla to meatus explains why meningeal and basilar conditions of necessity affect them both at the same time.

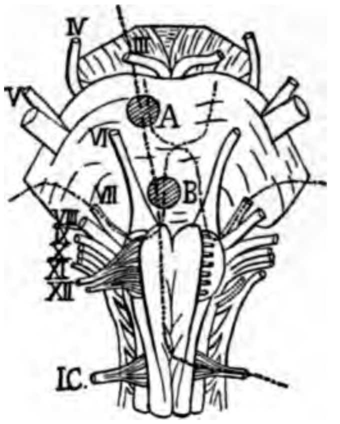

Fig. 44.—Diagram showing the course of facial and pyramidal fibers and the relations of cranial nerve-trunks. A, Lesion causing one-sided symptoms; B, lesion causing crossed paralysis of the face on one side and the limbs on the other.

The nucleus of the seventh receives fibers from the oculomotor nuclei above, which are destined to the orbicular muscle of the eyelids. By this mechanism the functions of winking, accommodation, and ocular movements are associated. Fibers from the hypoglossal nucleus below also pass to the nuclear centers of the seventh, and are eventually distributed to the orbicular muscle of the mouth, correlating the labial and lingual movements necessary in phonation, mastication, and other buccal processes.

The decussation of the seventh explains the phenomena of crossed or alternate paralysis of the face and limbs. A lesion in the pons above the decussation involves at once the seventh nerve and the pyramidal tract for the opposite side of the body, but below the facial crossing and above the pyramidal decussation a lesion involves the face on the same side and the limbs on the opposite side. Such a lesion must involve the lower third of the pons, approximately the portion below the superficial origin of the fifth pair.

After entering the internal auditory meatus the seventh nerve bends somewhat sharply, and presents a gangliform swelling, which receives the large superficial petrosal from the Vidian nerve containing the taste-fibers from the second branch of the fifth nerve by way of the sphenopalatine ganglion. The taste-fibers leave the facial nerve in the form of the chorda tympani after it has almost completely traversed the Fallopian canal, and, passing up through the tympanum, finally reach the anterior portion of the tongue with the lingual branch of the fifth. Within the Fallopian canal the facial gives off from within outward, first, near the ganglion of the knee, above mentioned, a motor branch to the tympanic plexus; second, a motor branch to the stapedius muscle; and, third, the chorda accompanied by a secretory branch to the salivary glands. The facial nerve, therefore, within the aqueduct contains (1) motor filaments for the facial muscles, (2) filaments of the special sense of taste for the anterior two-thirds of the tongue, (3) motor filaments for the internal ear, and (4) secretory fibers for the salivary glands. In addition there are a few filaments subserving common sensation for the external ear, derived from the fifth nerve, which pass with the facial to its exit.

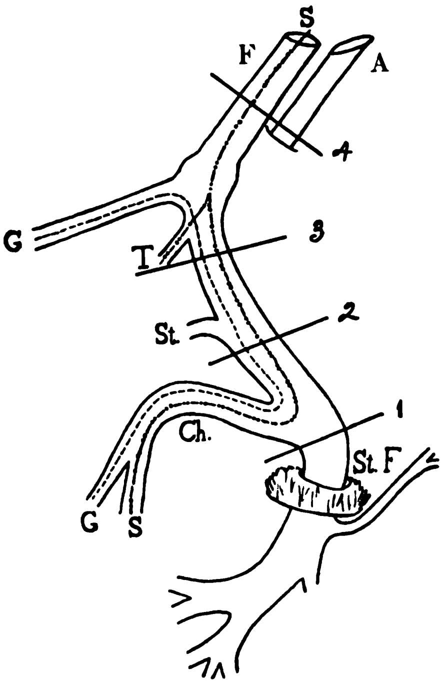

Fig. 45.—Diagram of facial nerve passing through the petron. F, Facial; A, auditory; St. F, stylomastoid foramen; Ch, chorda; S, S, salivary fibers; G, G, gustatory fibers; St, branch to stapedius muscle; T, tympanic branch.

After leaving the stylomastoid foramen the facial gives off (1) the sensory branches above referred to, (2) branches to the external auricular muscles, and (3) branches to the posterior portion of the occipitofrontalis. The trunk in the substance of the parotid then divides into (4) widely spread branches supplying motion to all the muscles of the face, to the platysma, the stylohyoid, and the posterior belly of the digastric.

Practically, the seventh nerve may be considered one of pure motion. It furnishes the trophic supply to the muscles of the face. The sensory and secretory filaments merely join it during its course and leave it before it reaches its periphery. When it is diseased the major symptoms are motor and consist of increased or decreased activity, giving rise to facial spasm or facial palsy as the condition is one of irritation or deficit. The muscles also waste. The addition of sensory and secretory disturbances enables us to locate the lesion with more or less exactness.

Spasmodic Affections of the Facial Nerve.—Facial Spasm.— Irritative lesions in the Rolandic face-centers may set up grimacing spasms on the opposite side, usually of an unrhythmical, clonic sort, which are not suggestive of any intention. Such a spasm may be the initial feature of a Jacksonian fit. Grimacing is the feature of all severe epileptic attacks that most impresses onlookers. In limited cortical lesions in the face-centers the spasm may be confined to a portion of the face, as to the angle of the mouth or to the eyelids and brow. These cortical spasms are usually attended by mental symptoms, such as an aura, confusion, or unconsciousness, and are practically independent of circumstances and surroundings. Feelings of apprehension and a distressed appearance commonly attend them. Their frequency is variable. Often they occur singly, at long and irregular intervals. An explosion, a sort of status, may take place so that the face is convulsed for many minutes or even hours, and then returns to a normal quietude, perhaps marked by a slight paresis or even by a distinct weakness, which is important and should always be sought.

The irritative lesion may be subcortical and produce the same conditions. At the nuclear level it would almost certainly involve other cranial nerves. Pontine and basilar growths have in rare instances been the cause of such facial spasms, and inflammatory invasion of the nerve-trunk is usually marked by twitchings in the muscles which subsequently present palsy. These twitchings often reappear with regeneration. All the above conditions produce a pure spasm.

The muscles supplied by the facial nerve, either alone or more commonly in conjunction with other groups, may be affected by spasmodic *tics*, including blepharospasm, mimic spasm, grimacing, etc. These are ordinarily the external manifestation of a fixed idea, and will be described under the psychoneuroses in Part VII.

Paralysis of the Seventh Nerve.—Facial palsy follows lesion of the seventh nerve at any point from the cortex to the stylomastoid foramen. In the face individual branches may be injured, with corresponding local motor loss. The most complete facial paralysis depends upon injury to the nerve-trunk. When it follows exposure to cold or occurs in rheumatic conditions it is often called *Bell's palsy*. The peripheral form, due to a lesion of the nerve-trunk after its entrance into the petron through the internal meatus, will first be considered, and then the nuclear and central varieties.

Peripheral Facial Palsy.—**Etiology.**—The facial nerve in the petrous canal is occasionally invaded by inflammation extending from the tympanic cavity. The separating plate of bone is often of extreme thinness, and may even be lacking, especially in children. Mastoid disease and pharyngeal inflammations have led to the same result. Fractures of the cranial base very frequently involve the petron, and may cause a facial palsy at once; or this may come on from inflammatory reaction after a few days, or later from the deposition of callus. In extremely rare instances hemorrhage within the canal produces paralysis almost instantly. In some infantile cases a cerebellar hemorrhage invades the canal with the same result. Injuries to the nerve at its foramen of exit, as by blows with sharp or blunt instruments and by the obstetrical forceps, may cause the palsy. In addition to the local disadvantage presented by the facial nerve being surrounded by a long bony passage, Neumann attributes much to the large number of lymphatics and lymphatic glands encircling it at its exit from the stylomastoid foramen. Stagnation of lymph here favors changes in the neighboring nerve-trunk. Upward extension of inflammation in the parotid or side of the neck into the canal is an occasional cause. By all odds the most common and usual cause is a neuritis due to cold, as by a draft of air on the side of the face during the night, or from an open window, or after prolonged outdoor exposure. The nerve-swelling to which this gives rise nips the nerve in its unyielding channel and paralysis supervenes.

Huebschmann,[1] from a series of 135 cases, found seventy-five per cent. to be of the so-called rheumatic form, nine per cent. due to ear disease, and six per cent. to injury. Most of the rheumatic cases occurred between the ages of ten and forty years, and males predominated about five to four. Reik [2] believes that the so-called rheumatic variety of facial palsy is very commonly dependent upon catarrhal or other inflammation of the middle ear, and urges an examination for otitis media in every case.

Any general depression of the physical state may act as a predisposing element. In this relation rheumatism occupies a chief place. Anemia, syphilis, tuberculosis, and alcoholism are also very favoring conditions, as is the puerperium. Syphilis may of itself cause a local lesion, but must at this location act very exceptionally. Gouty and leukocythemic neuritis may also involve the facial trunk directly. Sarbo[3] agrees with Neumann that there is commonly a personal predisposition to facial paralysis, and Arkwright[4] reports six cases in two families which indicate the same thing.

Symptoms.—*Motor.*—In a well-marked case, unless due to direct violence, within about twenty-four hours after the action of the inciting cause a little twitching in the affected side of the face is frequently noticed. Perhaps friends then first call attention to the distortion of the face, which in all expressional movements is drawn to the opposite side. The patient may first find that he is unable to expectorate with certainty, to whistle, or to puff out his cheeks and lips, owing to the loss of control over the labial positions. In two or three days the deformity is noticeable to all. The entire side of the face is affected, with the occipitofrontalis. Even the small muscles of the external ear on the same side are paralyzed. The unopposed muscles of the sound side draw the mouth in their direction, and the zygomatici elevate its angle. When the mouth is opened widely it presents an unsymmetrical outline. It is higher on the sound side and displaced to this side of the middle line. This may mechanically cause the projected tongue to deviate to the sound side, but if the mouth be passively held in a median position, the lingual curve disappears and full mobility of the tongue can be easily shown. The dorsum of the tongue is slightly depressed on the affected side, according to Schultze,[5] from weakness of the stylohyoid and digaster. Saliva and food accumulate in the flabby cheek and have to be displaced with the finger. Mastication, therefore, is mainly done on the sound side and, due largely to this fact, lateral furring of the tongue on the paralytic side may appear. Often there is some diminution of salivary flow on the paralyzed side, owing to the involvement of the secretory fibers which traverse the facial trunk. The nasolabial fold disappears on the injured side and is accentuated on the sound side. The chin and nose may deviate to the sound side. The eye on the paralyzed side can not be closed nor the eyebrow lifted. In attempts to close the eyes the eyeball on the affected side turns upward and can not be controlled. Winking is absent, and in the aged the lower lid often droops away from the ball. The cross-wrinkles on the forehead stop abruptly at the middle line.

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[1] "Neurol. Centralblatt," Nov. 15 and Dec. 1, 1894.

[2] "Johns Hopkins Hosp. Bull.," April, 1902.

[3] "Deut. Zeit. f. Nervenhl.," Mar., 1904.

[4] "Lancet," Jan. 23, 1904.

[5] "Münch. med. Wochens.," June, 1897.

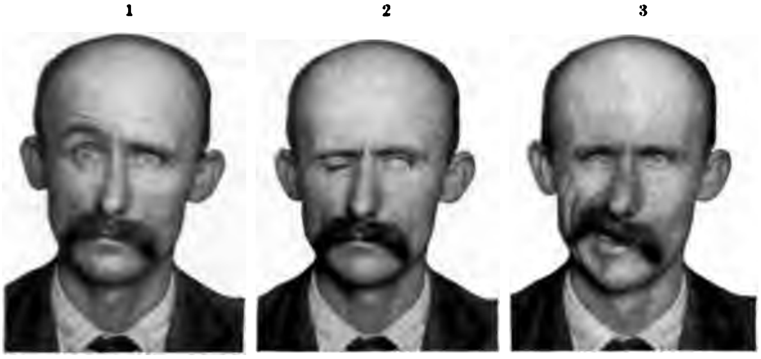

Fig. 46.—Facial palsy of left side. 1, Bilateral attempt to raise eyebrows; 2, bilateral attempt to close eyes; 3, smiling.

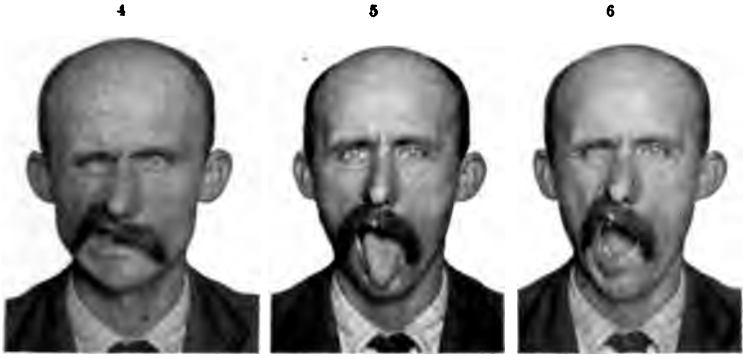

Fig. 47.—Facial palsy of left side. 4, forced effort to uncover teeth on both sides; 5, protruded tongue in middle line; 6, mouth open more widely on sound side in bilateral effort.

During moments of quiet little asymmetry may be apparent, but the slightest emotional or voluntary movement produces and accentuates all the motor symptoms. In young patients, where the expressional lines and wrinkles are absent or only slightly developed, the difference between the two sides is much less marked than in those of more mature years. The greater resilience of the skin and the larger adipose layer contribute to this effect. The *reaction of degeneration* is present in the muscles either in complete or modified degree. The muscle reflexes are abolished and McCarthy's supra-orbital reflex is lost. After a few weeks the *muscles waste*. The atrophic loss is not readily seen except in thin people. As the eye can not be closed, the exposed conjunctiva is often irritated and painful. At night it may become quite dry, and *conjunctivitis* of a severe grade may develop. From the relaxation of the lids the natural *flow of tears* to the lacrimal duct is interrupted and they overflow on to the cheek. In rare severe cases there is a herpetic eruption, but this is probably due to a simultaneous affection of the fifth nerve.

Disturbance of *common sensation* is slight or wanting. If in the first week the muscles of the cheek be grasped, some tenderness can be elicited, which is probably due to the irritation of the sensory twigs distributed to the degenerating facial branches. The *sense of taste* on the same side of the tongue in its anterior two-thirds, the part supplied by the chorda tympani, is often disturbed or abolished. Many patients during the first few days acknowledge subjective taste sensations of a peculiar sort; but ordinarily a careful search must be made in order to elicit the actual difficulty. *Hearing* is sometimes modified in such a way that low notes are more readily heard than on the sound side, while those of high pitch, as the ticking of a watch, are not distinguished with even ordinary acuteness. Middle-ear inflammation, if present, would be attended by some general loss of hearing and sometimes by tinnitus.

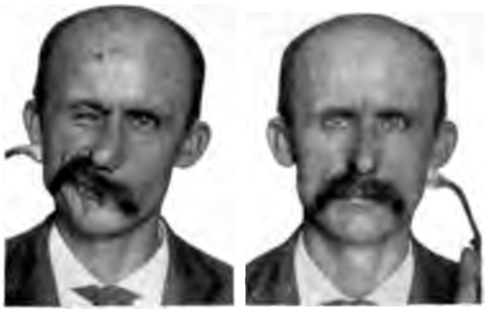

Fig. 48.—Facial palsy of left side. 7, Effect of faradism on sound side; 8, non-effect of same current on paralytic side.

In rare instances a *double facial palsy* is encountered. This usually is significant of intracranial disease, as pontine lesions, inflammation (especially of a syphilitic sort) at the base, or nuclear degeneration, which ordinarily involves other cranial nerves at the same time. A double otitis may produce it, or it may be part of a multiple neuritis, such as follows diphtheria and other general intoxications. In bilateral palsy the face hangs flabby, motionless, and without expression. The unwinking eyes and the drooping mouth give a vacuous look that is suggestive of dementia. All of the paralytic symptoms are present on both sides, but the distortions and asymmetry so prominent in the ordinary form are wanting.

Course.—The tendency of the palsy to improve in peripheral facial paralysis is marked, excepting in destructive lesions of the nerve-trunk. Cases that improve get practically well in from three weeks to eighteen months, depending upon the amount of injury done the nerve and the duration of pressure within the facial canal. This is true regardless of treatment in those cases that depend upon so-called rheumatic conditions, though improvement may without doubt be accelerated by proper management. The early loss of faradic stimulability of the muscles gradually passes away, but voluntary motion returns before the faradic current regains its control. Very frequently the paralyzed muscles will twitch or even act in vigorous spasm as regeneration in the nerve restores its irritability. Those cases which last a month or two, or more, usually leave traces for the rest of life. They may be so slight as to require careful scrutiny, or marked weakness may persist. Very exceptionally the palsy is permanent.

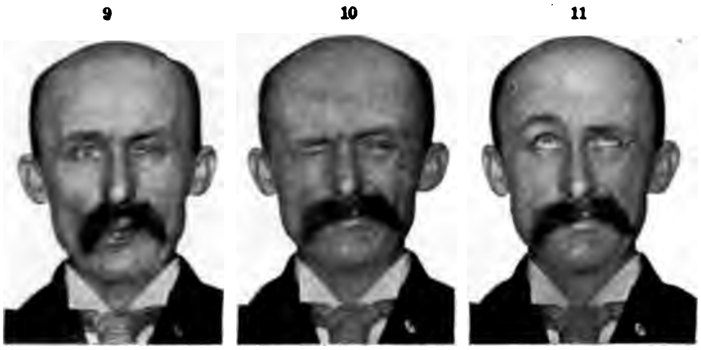

Fig. 49.—Same case six months later. 9 shows late contracture on the paretic side while the face is at rest; 10, contracture in the lower half of the face increased by gently closing the eyes, and at the same time shows weakness about left eye; 11, contracture increased by raising brows, showing overaction of zygomatici and weakness of frontalis on left side.

As the face recovers, in every instance the paretic side is likely to overact for all moderate voluntary bilateral movements. It would seem that the neuritis had left a certain nuclear irritability, so that a central influence, acting equally on both sides, produces a greater effect on the formerly paralytic muscles. This secondary *overaction* is most pronounced in the lower half of the face, and often leads the patient to suppose that the sound side is becoming paretic. When once strongly developed it practically remains for the rest of life, though slight degrees of it may pass away. While, therefore, the paralysis usually gets well, the sequel often remains and is irremediable, but is not of great amount or serious import. Indeed, many people who have never been affected show almost as much asymmetry of facial muscular action. Some cases present several attacks of facial palsy. Bernhardt[1] asserts that recurrence takes place in 70% of all cases and in men more commonly than in women. Second attacks are frequently associated with middle ear inflammation, syphilis, or diabetes. In the authors' experience second attacks are decidedly uncommon. Rossolimo[2] reports a recurring case associated with migrainous attacks.

Diagnosis.—The diagnosis of a facial palsy, if at all marked, is made at a glance in the early stage. Later on the secondary overaction may momentarily mislead, but its nature should be easily deciphered. In slight volitional movements the overacting and consequently formerly paralyzed side of the face responds in an exaggerated manner;

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[1] "Neurol. Centralblatt.," 1899.

[2] "Neurol. Centralbl.," Aug., 1901.

but if the muscular effort be forced, as in vigorously screwing up the eyes and in laughing, the paresis will be manifest in the lessened action of the orbicularis palpebræ, though at the same time the zygomatici on the same side may be strongly overacting and exaggerating the naso-labial furrow. In the same way a strong attempt to uncover the teeth will show a weakness on the affected side. The distinction between a cerebral and a peripheral palsy will be made clear in the consideration of lesions within the skull. It suffices here to say that disease of the cortex and in the brain above the nuclei, as in ordinary hemiplegia, produces no muscular atrophy, no reaction of degeneration in the muscle, and, with practical uniformity, never involves the brow and is most marked in the lower portion of the face. The tongue also is affected, as a rule. Lesions at the nucleus and in the pons almost invariably implicate other cranial nerves. Lesions at the base, as in meningitis, affect other cranial nerves usually, and always, when the face is paralyzed, produce deafness from injury of the more vulnerable auditory nerve, the parallel course of which with the facial has been pointed out.

The question as to the location and extent of the lesion in the usual peripheral palsy is an interesting one, and can be answered with some degree of precision. Referring to the diagram, page 118, it is evident that the neuritis, unless confined to a very short distance above the stylo-mastoid foramen, will (1) involve the chorda and (2) produce loss of taste on the same side of the tongue, with diminished salivation. A little higher (3) the tympanic tensor nerve is cut off and the auditory symptoms of hyperacousis for low tones, with blunting of hearing for notes of high pitch, will be added. At the internal (4) meatus the auditory nerve will also suffer and the chorda may escape.

Prognosis.—The prognosis, while very good for practical recovery in all cases of facial neuritis, should always be guarded and guided by an electrical examination, which, after the second week, often furnishes information of a precise nature. Simple slight cases present no diminution in faradic response or change in galvanic excitability. They may be expected to recover within a month. If galvanism and faradism of the nerve give diminished responses and galvanism of the muscles shows increased influence, with A. C. C. equal to or greater than C. C. C., the case will probably last two or three months. Finally, if the reaction of degeneration be complete, no responses in the nerve to either current, the muscles refusing to act to faradism and showing overexcitability to galvanism with the anode overpassing the cathode, a year to eighteen months must be allowed. In this last group of cases marked and persistent overaction may and should be foretold. Bordier and Fraenkel[1] insist that the outward and upward deviation of the eye when the patient tries to close the lids is proportionate to the severity of the nerve injury, and recovers in an equal ratio.

Traumata of the trunk of the nerve at its bony exit, even severing it, are not necessarily of grave outlook, as union and regeneration may follow. Middle-ear diseases and cranial fractures are likely to heal and the associated palsy to pass away. Naturally, the prognosis must be more guarded in these conditions, and is based largely upon the possibility of their recovery.

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[1] "Sem. Méd.," Sept., 1897.

Treatment.—The tendency to recovery being pronounced, can we shorten the duration of the disability? It seems probable. If the case is seen early, a blister or leech on the mastoid may reduce the congestion in the facial canal. Hot applications to this region may be tried. If not thoroughly and persistently used, they are worse than useless and later on quite valueless. When the case is developed we recognize that the lesion of the trunk shuts off trophic and motor control. The indication is to maintain the muscles in as perfect a condition as possible until the way for nuclear and cortical influence is reopened. Further, we should strive to keep the muscles as responsive as possible to the slight fraction of motor control that remains, or, if none is present, to the weak and inefficient impulses that will first reach them. To this end there are three things to do: (1) Keep the face in place by having the patient constantly push the cheek and mouth to their proper positions, from which every smile and word cause them to be dragged by the sound muscles. In the same way have the eyelids rubbed over the eyeball frequently, and at night a compress should be worn to keep them closed. This has the additional advantage of diminishing and often of preventing conjunctivitis. A weakened muscle stretched by its sound antagonists is not only placed at a mechanical disadvantage, but actually injured in its nutrition. (2) By means of massage and hot douches accelerate and improve circulation, and thereby the nutrition of the affected muscles. (3) By electrical stimulation of the muscles, commenced from the first day, keep up their responsiveness. For this purpose a galvanic current only is needed. Faradism to contract the muscles, or the faradic brush, are needless and painful. Ordinarily the best plan is to use the anode at the motor points on the side of the face and to daily systematically cause the muscles to gently contract six to ten times by anodal closures. This pole is the least painful and the most active in the presence of degeneration.

Traumatic cases are to be treated on surgical lines. If the nerve-trunk is divided, an attempt should be made to suture it. Breauvoine,[1] in the manner advocated by Faure and Furet, reports a fair success following the establishment of an anastomosis between a diseased facial and the spinal accessory. This method is only applicable to those cases in which the nerve has no other possibility of regeneration. Anastomosis with the hypoglossal promises even better results.[2] Tympanic, mastoid, and pharyngeal trouble will require topical measures. When the neuritis is a part of general multiple neuritis, attention is directed to the toxic cause. Guided by the rheumatic idea, such remedies as the salicylates have been recommended and may be tried if the observer can convince himself that such a blood-state exists. Anemia and diminished physical forces from any general cause are not to be neglected. Strychnin may be of some service, acting as a nuclear stimulant and general tonic.

Nuclear palsy of the seventh nerve manifests itself by the same peripheral motor distribution as that found in diseases of the trunk, but lacks the gustatory, salivary, and sensory disturbances. The muscles degenerate in the same way and show the same electrical conditions. Almost invariably other adjacent cranial nuclei are implicated. This gives rise to a symptom group that easily differentiates the nuclear palsy from the peripheral form. In bulbar palsy the facial nucleus is generally invaded, and it may be affected in polio-encephalitis superior. The nuclear lesion usually produces bilateral symptoms. The pyramidal tracts are also commonly affected, and symptoms in the trunk, and especially in the extremities, are present.

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[1] "Travaux de Neurologie Chirurgicale," Jan., 1901.

[2] Ballance and Stewart, "Br. Med. Jour.," May 2, 1903; Taylor and Clark, "Med. Rec.," Feb. 27, 1904.

An alternating palsy of the face on one side and of the limbs on the other is due to a pontine lesion (see Fig. 44) affecting the facial fibers before their crossing and the pyramidal tract above its medullary decussation.

Supranuclear facial palsy results from any lesion destroying the cortical facial centers or interrupting the communication of these centers with the facial nucleus. Ordinary capsular hemorrhage is, therefore, a frequent cause of facial palsy, but almost invariably at the same time causes hemiplegia. In these supranuclear palsies, as has already been indicated, the upper portion of the face partially or entirely escapes. Particularly is this true of the orbicularis palpebrarum, which acts voluntarily. The slight weakness that is present is often shown by a partial ptosis completely at variance with the constantly open eye of the peripheral palsy, but the eye on the affected side usually can not be closed as vigorously as the other. All expressional bilateral movements in the lower face may be fairly retained, while voluntary control is practically lost. In the peripheral form the loss is equal in both varieties of action.

In supranuclear palsy the muscles respond actively and normally to electricity, and their trophic condition is not impaired. In other words, the lower neuron is not involved. The reflexes are present for the same reason, and there are no auditory, secretory, or gustatory symptoms. A lesion that involves the optic thalamus may, according to Bechterew, cause loss of emotional expression on the opposite side. A lateral loss of expressional movements, with the retention of voluntary motion, therefore, points to the optic thalamus, which contains the centers for emotional expression. This loss has been found associated with corresponding bilateral hemianopsia resulting from the same lesion.

CHAPTER VIII.

DISEASES OF THE EIGHTH CRANIAL NERVE.

Anatomical Considerations.—The eighth cranial nerve is purely sensory. It is made up of two portions: the cochlear branch, which alone conducts sound impressions, and the vestibular branch, which conducts space sensations from the semicircular canals. An accessory and, so to speak, adventitious portion is the intermediate nerve of Wrisberg, which is vasomotor and secretory in function. It passes to the facial nerve and finally controls salivation. In consistence the auditory stem is much less firm than the facial. It follows that basilar lesions which affect the seventh almost necessarily involve the softer auditory, which lies beside it. But, on the other hand, a lesion that injures the auditory may not affect the more resisting facial or portio dura.

At the apparent origin of the auditory trunk the internal vestibular portion, the one related to equilibration, passes backward and inward between the restiform body of the medulla and the ascending root of the trifacial nerve, to terminate in two groups of large cells. The outer cochlear portion, the true auditory root, separates from the vestibular root near the apparent origin, and, passing outward around the restiform body, turns inward on the floor of the fourth ventricle as the striæ acusticæ, which dip down in the middle line to the nuclear cells of Clarke. At the point where these branches diverge the cochlear root presents a group of cells, for the vasomotor root of Wrisberg, analogous to a posterior root ganglion. Another group of cells, the acoustic tubercle, often of large size in animals, is placed just outside the restiform body, and is considered a pure auditory nucleus. The higher relations of the auditory nuclei are not clearly known, nor the exact course of the radiations to the cortex. There is every reason to believe that the vestibular nuclei are in relation with the cerebellum and with the cerebrum. The cochlear nuclei are connected with the temporosphenoidal cortex by fibers which pass through the posterior third of the sensory division of the internal capsule. Each ear is represented on both sides of the brain, but also principally upon the opposite side. The auditory center for speech, however, in right-handed individuals is practically confined to the left temporal lobe, the destruction of which produces word-deafness, or a loss of spoken-word memories. At its peripheral termination the auditory nerve enters the cribriform opening in the internal meatus. The auditory portion is distributed to the cochlea and the organ of Corti. The labyrinthian portion is distributed to the vestibule and ampullæ of the semicircular canals.

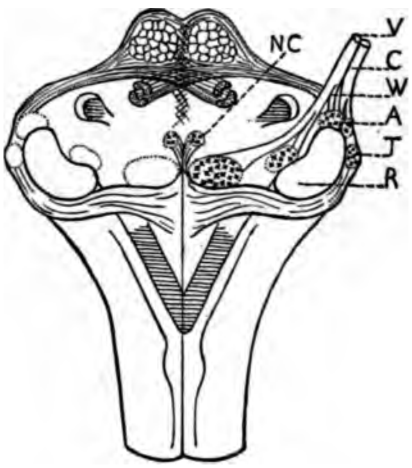

Fig. 50.—Diagram of a section showing the origin of the eighth cranial nerve. V, Vestibular portion; C, cochlear portion; W, accessory of Wrisberg; A, accessory nucleus; T, acoustic tubercle; R, restiform body; NC, nucleus of Clarke (after Brissaud).

Irritation of the auditory portion of the eighth nerve is marked by *auditory hyperesthesia*, by increased acuteness of hearing (*hyperacousis*), and by *tinnitus*. It must be understood that continued irritation of the auditory apparatus may result in diminished or lost function, just as overstimulation of any tissue or organ finally produces weakness and involution. It follows that tinnitus, for example, is often found with defective hearing. *Hyperesthesia* is frequently present in excitable and nervous persons. Migraine and all forms of pain often render the sufferer more sensitive to sudden noises, especially of high pitch, but actual hyperacousis is rare. In meningitis, acute mania, and under the influence of some stimulant drugs, such as alcohol, opium, anesthetics, Indian hemp, and caffein, the special sense of hearing is at times actually exalted. It is also conceivable that an irritant lesion of the auditory cortical centers might give rise to hyperacousis, and perhaps this is also the explanation of some hallucinations of hearing and sometimes of the rare auditory auræ of epilepsy. The increased acuteness of hearing for low tones in facial palsy has already been described.

Irritation or disease of any part of the auditory mechanism is likely to produce a tinnitus which may vary widely with the same and with different cases. It may resemble hissing, roaring, buzzing, singing, ticking, throbbing, the sound of bells, and every conceivable monotonous noise. Cerumen or foreign bodies in the external meatus, inflammation of the middle ear, Eustachian occlusion, the throbbing of the carotid in its canal in the petron, rhythmical clonic spasm of the palate and orifice of the Eustachian tube in hysterics, meningeal irritation of the auditory nerve-trunk, perforations of the drum-head, sclerosis of the internal ear, and many other conditions may be symptomized by a tinnitus aurium. A tinnitus is present in some cases where aurists are unable to detect any peripheral cause, and persists practically unchanged throughout life. As a rule, a continuous tinnitus interferes with hearing, which is thereby diminished. Fortunately, it is frequently unilateral. Certain drugs, as quinin and the salicylates, cause tinnitus, and quinin in large and repeated doses has caused permanent deafness. A careful aural examination is needed in every case of tinnitus, and the reader is referred to the special works on ear diseases. The treatment is that of the inciting condition.

Paralysis of the auditory nerve and unilateral deafness may follow a cortical lesion involving the temporal lobe. Memories of heard speech seem to be stored up in the first left temporal convolution. A destructive lesion here is followed by *word-deafness*, and the patient no longer understands what is said, though he clearly hears the tones of the voice and all sounds. A bilateral lesion of the temporal cortex causes complete deafness. Lesions in the sensory portion of the capsule may cause a unilateral deafness on the same side with the accompanying hemianesthesia. A unilateral loss or diminution of hearing in the hemianesthesia of hysteria is not uncommon. It may come on suddenly and disappear in the same manner. The presumption is that the cortex is functionally at fault. Complete—that is, bilateral—hysterical deafness is seldom encountered. Nuclear disease of the eighth nerve is practically unknown, though tumors of the restiform body [1] and the medulla may involve the cochlear root.

According to Virchow, the auditory trunk, most frequently of all the cranial roots, is the seat of new growths. Tumors at this point give rise to the general symptoms of brain-tumors, but are capable of definite diagnosis in at least some instances. According to Fraenkel and

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[1] Brissaud, "Leçons sur les Maladies Nerveuses," Paris, 1895.

Hunt,[1] who have collected nine cases, the symptoms may be arranged as follows: (1) General: Headache, vertigo, vomiting, optic neuritis, bradycardia. (2) Focal: Peduncular ataxia, cerebellar ataxia, lateropulsion, hemi-asynergy, homo-contralateral and crossed paralyses of the extremities, paralysis of the basilar cranial nerves, especially the seventh, sixth, and fifth, dysarthria, dysphagia, nystagmus, paralysis of the conjoint movements of the eyes, inequality of the pupils, and attacks of Adam-Stokes syndrome. (3) Special: Serious impairment of the function of the auditory nerve, of long standing and gradual onset. Gouty deposits and hemorrhage in the substance of the root have been found. It very frequently is involved in syphilitic meningitis. In purulent meningitis infection may travel along its sheath into the inner ear and produce deafness that is usually permanent. After exposure to cold the eighth nerve is sometimes subject to a neuritis similar to that so common in the facial, which may or may not be associated in the process. The condition is marked by diminished or complete loss of hearing, but is of favorable prognosis. Artisans who work constantly amid loud noises—as boilermakers, tinsmiths, and other metal workers, engine-drivers and firemen on railroads—often present a partial loss of hearing that may progressively increase. They sometimes hear better in the accustomed turmoil than in quiet places, and this is also true, but much less commonly, of ordinary deafness. The auditory nerve may be injured within the petron by the extension of inflammation from the mastoid, tympanum, or pharynx, and by basilar fractures.

Diagnosis.—The first thing to be settled in a case of deafness is the integrity of the nerve. If the nerve is at fault, the condition is called *nervous or nerve deafness.* When there are no basilar symptoms, involvement of other cranial nerves, bulbar or cerebral indications, and when sounds of high and low pitch are not heard through the air or by bone-conduction, we may safely locate the disease in the nerve. Rinne's test (see p. 64) enables us, when the hearing is reduced, to fairly determine whether the difficulty is in the conducting apparatus or in the nerve. A great reduction of hearing, in which air-conduction remains better than bone-conduction, but in which both are deficient, points to nervous deafness. Lesions within the brain-stem and in the temporal lobe must be determined by the association of symptoms peculiar to these localities.

Treatment.—In the treatment of nervous deafness we have first to investigate the aural apparatus and remove, if possible, any diseased conditions that may be present. All acute inflammatory trouble must subside before active measures are instituted. The use, then, of strychnin, preferably hypodermatically, in much the same way as for optic atrophy, can be recommended. Electricity has small claims to notice, though usually suggested. Unfortunately, very little improvement can be expected. Nerve deafness of sudden onset, whether due to syphilis, neuritis, congestion, or hemorrhage into the internal ear, is sometimes favorably modified by the use of pilocarpin in full doses twice daily for a week or two.[2] Free action on the skin is to be produced. In these cases quinin and the salicylates are contraindicated, though sometimes of value in the chronic forms.

[1] "Ann. of Surg.," Sept., 1904.

[2] Dundas Grant, "Brit. Med. Jour.," Nov. 16, 1896.

Irritation of the Vestibular Portion of the Eighth Nerve—*Aural Vertigo, Ménière's Disease.*—The function of the semicircular canals is still subject to dispute. It can at least be accepted that their irritation or injury may cause vertigo and disturbance of equilibration. Recognition of relations to space and orientation undoubtedly are subserved by them to a considerable degree. Their innervation is by the vestibular portion of the eighth cranial nerve. It has long been recognized that disturbance in the internal or middle ear, and even in the external meatus, may cause not only tinnitus, but vertigo. The sudden inflation of the drum through the Eustachian passage, the use of interrupted galvanic currents about the ear, and any instrumentation within the tympanum may produce giddiness. The only essential common character of all these causes is that they produce irritation. If destruction of the vestibular branch takes place, the vertigo usually ceases. In some cases of aural vertigo due to middle- or internal-ear disease, external objects seem always to revolve to the right or to the left. In other instances the vertigo is subjective and the patient feels as if revolved to the right or left or as if falling forward, backward, or downward. The intensity of the vertigo varies greatly. It may be quite insignificant or it may be so pronounced that the patient holds to any neighboring person or object, staggers, or is even forcibly projected in some given direction. The attacks are usually paroxysmal, with relative freedom in the intervals. In the form described by Ménière the victim is struck down as if shot, there may be unconsciousness for a few moments, and the patient is often pale and covered with perspiration. Nausea and vomiting may occur.

The great majority of cases of aural vertigo occur after thirty years of age. In childhood they are extremely infrequent and rapidly increase after middle life, men being affected twice as frequently as women. Gout, rheumatism, and the sclerotic changes of old age are frequently at the bottom of the symptoms. These may act directly upon the labyrinth or indirectly through the blood-supply and the pressure of the endolymph. A vasomotor element is given considerable importance by some. The irritation of the vestibular filaments is usually associated with auditory phenomena, so that tinnitus and defective hearing are almost invariably present. The close anatomical relations of the two portions of the eighth nerve explain this. In some cases the labyrinth has been found the seat of hemorrhage or local disease. Sometimes its epithelial structure is degenerated. Usually disease of the middle and external ear is wanting in the severest cases, while decreased hearing and tinnitus point strongly to involvement of the nerve itself. Some of the cases of the Ménière type show a progressive tendency, with failing hearing, first in one, then in the other ear. Complete physical disability through the vertigo and attacks of falling may ensue. In some instances the disease remains at a standstill for years, and may even recede and hearing be restored. Again, when hearing is lost the vertiginous attacks may cease. Milder varieties run various courses, depending upon their causation and other conditions.

Diagnosis of an aural vertigo depends in practice mainly upon the association of auditory symptoms. Tinnitus or defective hearing, or both, are ordinarily present. The defect in hearing, as tested by bone-conduction, is sometimes unexpectedly great, the ticking of a watch when placed on the mastoid being inaudible. If the vertigo is produced or increased by changing the air-pressure in the tympanum, as by firmly pressing the tragus into the meatus or by Politzerization, the significance of that fact is great. Frequently the attack of vertigo is associated with an intensification of the tinnitus, or there are subjective sounds of a violent character, described as "pistol shots," "something breaking in the head," etc. Sometimes a quick movement of the head in a given direction produces it. This apparently has relation to a particular semicircular canal, which is mainly or alone affected. A further characteristic of aural vertigo is the fact that the subjective or objective gyrations are uniform in the given case, or the stagger or falling is always in the same direction. In epilepsy we not infrequently encounter an indescribable vertiginous aura, but never the formulated vertigo of aural disease. The epileptic attack is usually followed by mental hebetude, which is lacking in aural attacks, where the vertigo may be maintained for a long time, giving rise to distressed feelings, vomiting, and collapse. The sensorium is always clear in aural vertigo, excepting the initial momentary unconsciousness of the severest form, or in the delirium that a continuation of the extremest variety may produce in very rare instances. The persistent vomiting usually gives rise to the idea of "biliousness," and frequently a brisk cathartic, relieving all the symptoms of vertigo, is supposed to confirm the idea of intestinal or hepatic derangement, its influence on cerebral circulation being overlooked. Again, the sudden onset of the attacks in the Ménière form suggests cerebral disease or cardiac attacks, to which mistake the age of the patient and his arterial degeneracy often conduce. The repetition of the aural attack during periods of rest, and even during sleep, with absence of cardiac and cerebral symptoms in the meanwhile, will correct a misconception of this character.

The labyrinthine variety may be readily confused with ocular vertigo in some instances, as it occasionally gives rise to nystagmus, and even has produced diplopia. The patients sometimes describe oscillating movements in viewed objects, rapid in one direction with slow return, similar to the nystagmic movements of the eyeballs. The mutual dependence of space sensations and ocular impressions only needs to be mentioned to explain the secondary ocular movements. Ocular vertigo ceases the moment the eyes are closed, but this has no effect on the aural form. Vertigo is associated with numerous abdominal disturbances, particularly those of the stomach, liver, and small intestine. These forms of vertigo are usually attended by indigestion or other symptoms of a local character, and the vertigo lacks the distinctive gyratory feature of ear trouble. In some cases of aural vertigo, however, the patients complain merely of "dizziness," "giddiness," or "swimming" sensations. If the vestibule alone is involved, without any implication of the cochlea, as is conceivable, all auditory symptoms default. In such cases the diagnosis must largely depend on the exclusion of other sources of vertigo.

Treatment.—If aural vertigo is recognized as an irritation symptom,

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The great majority of cases of aural vertigo occur after thirty years of age. In childhood they are extremely infrequent and rapidly increase after middle life, men being affected twice as frequently as women. Gout, rheumatism, and the sclerotic changes of old age are frequently at the bottom of the symptoms. These may act directly upon the labyrinth or indirectly through the blood-supply and the pressure of the endolymph. A vasomotor element is given considerable importance by some. The irritation of the vestibular filaments is usually associated with auditory phenomena, so that tinnitus and defective hearing are almost invariably present. The close anatomical relations of the two portions of the eighth nerve explain this. In some cases the labyrinth has been found the seat of hemorrhage or local disease. Sometimes its epithelial structure is degenerated. Usually disease of the middle and external ear is wanting in the severest cases, while decreased hearing and tinnitus point strongly to involvement of the nerve itself. Some of the cases of the Ménière type show a progressive tendency, with failing hearing, first in one, then in the other ear. Complete physical disability through the vertigo and attacks of falling may ensue. In some instances the disease remains at a standstill for years, and may even recede and hearing be restored. Again, when hearing is lost the vertiginous attacks may cease. Milder varieties run various courses, depending upon their causation and other conditions.

Diagnosis of an aural vertigo depends in practice mainly upon the association of auditory symptoms. Tinnitus or defective hearing, or both, are ordinarily present. The defect in hearing, as tested by bone-conduction, is sometimes unexpectedly great, the ticking of a watch when placed on the mastoid being inaudible. If the vertigo is produced or increased by changing the air-pressure in the tympanum, as by firmly pressing the tragus into the meatus or by Politzerization, the significance of that fact is great. Frequently the attack of vertigo is associated with an intensification of the tinnitus, or there are subjective sounds of a violent character, described as "pistol shots," "something breaking in the head," etc. Sometimes a quick movement of the head in a given direction produces it. This apparently has relation to a particular semicircular canal, which is mainly or alone affected. A further characteristic of aural vertigo is the fact that the subjective or objective gyrations are uniform in the given case, or the stagger or falling is always in the same direction. In epilepsy we not infrequently encounter an indescribable vertiginous aura, but never the formulated vertigo of aural disease. The epileptic attack is usually followed by mental hebetude, which is lacking in aural attacks, where the vertigo may be maintained for a long time, giving rise to distressed feelings, vomiting, and collapse. The sensorium is always clear in aural vertigo, excepting the initial momentary unconsciousness of the severest form, or in the delirium that a continuation of the extremest variety may produce in very rare instances. The persistent vomiting usually gives rise to the idea of "biliousness," and frequently a brisk cathartic, relieving all the symptoms of vertigo, is supposed to confirm the idea of intestinal or hepatic derangement, its influence on cerebral circulation being overlooked. Again, the sudden onset of the attacks in the Ménière form suggests cerebral disease or cardiac attacks, to which mistake the age of the patient and his arterial degeneracy often conduce. The repetition of the aural attack during periods of rest, and even during sleep, with absence of cardiac and cerebral symptoms in the meanwhile, will correct a misconception of this character.

The labyrinthine variety may be readily confused with ocular vertigo in some instances, as it occasionally gives rise to nystagmus, and even has produced diplopia. The patients sometimes describe oscillating movements in viewed objects, rapid in one direction with slow return, similar to the nystagmic movements of the eyeballs. The mutual dependence of space sensations and ocular impressions only needs to be mentioned to explain the secondary ocular movements. Ocular vertigo ceases the moment the eyes are closed, but this has no effect on the aural form. Vertigo is associated with numerous abdominal disturbances, particularly those of the stomach, liver, and small intestine. These forms of vertigo are usually attended by indigestion or other symptoms of a local character, and the vertigo lacks the distinctive gyratory feature of ear trouble. In some cases of aural vertigo, however, the patients complain merely of "dizziness," "giddiness," or "swimming" sensations. If the vestibule alone is involved, without any implication of the cochlea, as is conceivable, all auditory symptoms default. In such cases the diagnosis must largely depend on the exclusion of other sources of vertigo.

Treatment.—If aural vertigo is recognized as an irritation symptom, its rational treatment will depend on appreciating and, if possible, removing the basic disease. Cases may be relieved or even cured by Politzer's inflation, by the removal of cerumen, or by the correction of a pharyngeal catarrh. In others the sclerotic changes in the labyrinth are irremediable and treatment is directed to reducing the irritability by bromids. Charcot strongly recommended in the Ménière type the use of quinin in large doses, but others have not had his success with that drug, and it should not be used in acute cases. He even advocated the destruction of the inner ear, producing loss of hearing, or, in other words, a removal of all irritability and the cessation of the vertigo at once, in the same way that sometimes occurs naturally. This has been practically applied by Milligan [1] with favorable results in three cases. Removal of the malleus and incus and mobilization of the stapes have given relief in many instances and may be advised with propriety, especially if the hearing is greatly impaired. Gout and arteriosclerosis, middle-ear disease, and lesions of the auditory stem must be treated in their own several ways. Electricity is of questionable value, though it is asserted by some that the positive pole over the tragus and the negative on the back of the neck, with a current of three or four milliamperes gradually increased from zero, continued for five minutes and then decreased, has a quieting influence. All interruptions should be avoided. Cases of acute onset are sometimes benefited by pilocarpin, as in nervous deafness, with which they are usually combined.

CHAPTER IX.

DISEASES OF THE GLOSSOPHARYNGEAL, VAGUS, AND ACCESSORY NERVES.

Anatomical Considerations.—The glossopharyngeal and pneumogastric nerves and the bulbar portion of the spinal accessory should be considered as one mechanism. Their nuclei in the medulla are practically inseparable, and they continuously furnish sensation and motion to the gastro-intestinal tract from the pharynx to the duodenum. In addition they supply motor filaments, which all come from the accessory portion, to the lungs, larynx, and heart. They are visceral nerves. It is to be kept clearly in mind that the spinal portion of the accessory is a pure motor nerve to the skeletal muscles of the neck, and is only locally associated with the pneumogastric. The interrelations of the glossopharyngeal-vagus-accessory group are so complex, their distribution so wide-spread, and their indirect disturbances so vague that they furnish many perplexities. A short outline of the glossopharyngeal is first given, and then the vagus and true accessory are discussed together.

DISEASES OF THE GLOSSOPHARYNGEAL NERVE.

The ninth cranial nerve is still a source of anatomical contention and physiological doubt. In consequence its diseased conditions are uncertain and obscure. Practically, in man, it is never alone diseased. If its relations to other cranial nerves are considered, this fact is readily understood. Through Jacobson's nerve it forms, with the sympathetic, the tympanic plexus, whence a branch connects it through the Vidian with the facial nerve, and another branch through the small superficial petrosal connects it with the otic ganglion. It is connected with the pneumogastric at the petrous ganglion of that nerve, and also in the pharyngeal plexus. Its nuclei are intimately associated with those of the vagus and accessory nerves. It probably subserves sensation in the upper part of the pharynx and in the tympanum, and nausea is associated with its disturbance. Probably through its distribution to the root of the tongue it peripherally carries the fibers of the special sense of taste for that area, but these are not embraced in its root. They reach the brain by a circuitous route, probably entering the petrous ganglion of the glossopharyngeal nerve from the middle branch of the fifth through the tympanic plexus and otic ganglion. It seems to have some motor control of the upper portion of the pharynx, and, perhaps, of the palate.

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[1] "Brit. Med. Jour.," Nov. 5, 1904.

Intracranial disease and cranial fractures may implicate the glossopharyngeal, causing weakness and some insensitiveness in the upper pharynx and in the palate. Its nuclei in the medulla usually suffer in bulbar palsy, and thus are produced, at least in part, the pharyngeal symptoms of that disease.

DISEASES OF THE VAGUS AND BULBAR PORTIONS OF THE ACCESSORY.

Pharyngeal Branches.—The pharyngeal branches of the pneumogastric follow below the glossopharyngeal, and with it form the pharyngeal plexus, supplying motion and sensation to the uppermost portion of the intestinal tube. These branches are paralyzed by nuclear disease and in diphtheric palsy, but seldom otherwise. Bulbar involvement is invariably attended by symptoms in other cranial nerves. The pharynx is more or less insensitive and motionless. The pharyngeal reflex is lost. Food tends to accumulate and lodge in the gullet or overflows into the larynx, producing spasmodic cough and strangling. If the palate at the same time is weakened, food and fluids may be forced into the nasal passages and regurgitate through the nose. A pharyngeal spasm furnishes the condition commonly noted in hysteria as "globus," or esophagismus, and is always functional. At times it may be mistaken for pharyngeal paralysis, or the difficulties in swallowing in the latter may be attributed to spasm. The use of a sound will at once clear the doubt. The decided pharyngeal grasp of health is increased in spasm and lost in paralysis. Moreover, spasm is temporary or recurrent, and paralysis is continuous.

Laryngeal Branches.—The larynx is innervated by two branches of the pneumogastric: (1) The *superior laryngeal* governs the movements of the epiglottis and controls tension in the vocal cords through the cricothyroid, which is the only intrinsic laryngeal muscle supplied by this nerve. It also furnishes sensation to the larynx above the vocal cords. (2) The *recurrent laryngeal,* which turns about the aorta on the left side and the subclavian artery on the right side, supplies sensation to the trachea and to the larynx below the vocal cords. It controls all the intrinsic laryngeal muscles except the cricothyroid. These muscles have three principal vocal actions: First, to draw the vocal cords tense; second, to bring them close together; third, to draw them apart. Though many laryngeal movements are highly complex, requiring the synergic action of several groups of muscles, it is well to remember that the *chief tensors* are the cricothyroids, the *chief abductors* are the posterior crico-arytenoids, the *chief adductors* are the lateral crico-arytenoids. In addition, the thyro-arytenoids, which in part form the vocal cords, serve to stiffen them and make their apposition uniform and effective. By some they are considered tensors and by others laxors of the cords, and probably serve both purposes.

Laryngeal paralyses vary in degree and in distribution. They may be unilateral or bilateral, partial or complete. Further, the abductors, the adductors, or the tensors of the cords may be alone or mainly involved. Abductor paralysis is, however, by far the most common,[1] even when the lesion falls upon the recurrent. A full knowledge of the anatomy and mechanism of the larynx is required to understand this subject, and the use of the laryngoscope is requisite for exact diagnosis. The following table is given to show the common varieties of laryngeal paralysis, with diagrams of the corresponding mirror pictures, which should be compared with the normal outlines in phonation, respiration, and death.

LARYNGEAL PARALYSES.

FORM OF PARALYSIS.	MUSCLES INVOLVED.	CAUSES.	SYMPTOMS.
Bilateral adductor paralysis. (Fig. 54.)	Both lateral crico-arytenoids and the arytenoideus.	Anemia, physical weakness, hysteria.	Voice lost, but cough and laugh phonetic; respiration and swallowing normal; no pain.
Unilateral adductor paralysis. (Fig. 55.)	One lateral crico-arytenoid.	Toxemia, lead, diphtheria, cerebral disease, small-pox, syphilis, phthisis.	Diminished voice; hoarseness; coughing, laughing, and sneezing diminished; difficulty in swallowing occasional.
Bilateral abductor paralysis. (Fig. 56.)	Both posterior crico-arytenoids.	Toxemia, hysteria rarely, injury to both recurrent nerves, as by enlarged bronchial glands; diseased thyroid, new growths in the neck, etc.	Voice little affected for ordinary efforts; respiration impeded; extreme inspiratory stridor, with free expiration.
Unilateral abductor paralysis. (Figs. 57 and 58.)	One posterior crico-arytenoid.	Stretching or injury to one recurrent nerve, as by aortic aneurysm, and the same causes as in the bilateral form, acting on one side only.	Voice harsh, impure, and hoarse; some inspiratory stridor.

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[1] Semon, "Brit. Med. Jour.," Jan. 1, 1898.

LARYNGEAL PARALYSES.—(*Continued.*)

FORM OF PARALYSIS.	MUSCLES INVOLVED.	CAUSES.	SYMPTOMS.
Bilateral paralysis of tensors.	Cricothyroids.	Colds; voice straining; diphtheria.	Hoarseness; inability to take high notes.
Paralysis of the cords proper. (Figs. 59 and 60.)	Thyro-arytenoids.	Overexertion, hysteria.	Loss of falsetto notes and uncertainty of voice-production; usually attended by some adductor paresis, and frequently by loss of power of the arytenoideus.

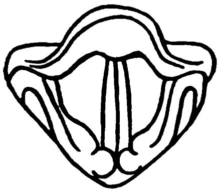
Fig. 51.—Normal phonation.

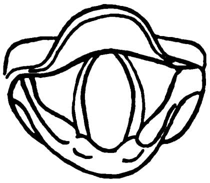
Fig. 52.—Normal deep inspiration.

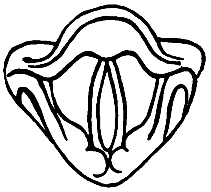
Fig. 53.—Normal cadaveric position.

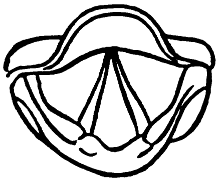
Fig. 54.—Bilateral adductor paralysis. Attempted phonation.

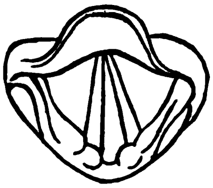
Fig. 55.—Left adductor paralysis. Attempted phonation.

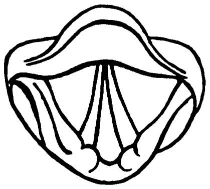
Fig. 56.—Bilateral abductor paralysis. Deep inspiration.

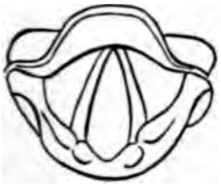
Fig. 57.—Left abductor paralysis. Deep inspiration. Affected cord in cadaveric position.

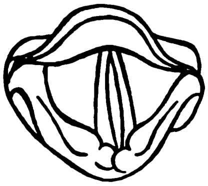
Fig. 58.—Left abductor paralysis. Phonation. Affected cord in cadaveric position. Right cord crossing median line.

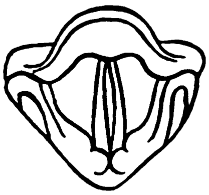
Fig. 59.—Bilateral thyro-arytenoid paralysis.

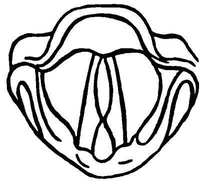
Fig. 60.—Bilateral thyro-arytenoid paralysis and paralysis of arytenoideus, giving an hour-glass opening.

In complete bilateral paralysis of the larynx, such as results from injury to both recurrent nerves, the vocal cords occupy the cadaveric position, and are motionless. There is no voice, and coughing and sneezing are impossible. Deep inspiration develops stridor. If the *complete paralysis is unilateral,* the motionless, paralyzed cord occupies the cadaveric position, while its fellow moves actively in phonation and respiration, even passing the middle line in adduction. The voice is low-pitched and hoarse, cough is absent, and stridor only appears on very deep inspiratory efforts.

The treatment of laryngeal palsies depends on that of the causative condition. Laryngeal palsies due to surgical injury of the nerve, as by the inclusion of the pneumogastric in ligation of the carotid, or its division in operations on the thyroid, have occurred. Here the immediate danger is to the heart. In the diphtheric forms and in other toxic varieties, the use of electricity is recommended. To be of any value it must be applied with the intralaryngeal electrode and requires special skill. The neuritic forms of laryngeal palsy are of fair prognosis, with or without treatment, if the patient survives the early effects of the toxemia. The nuclear varieties are practically irremediable.

Anesthesia of the larynx is occasioned by interference with the superior laryngeal nerve, which supplies sensation above the vocal cords. It may be unilateral or double-sided. In hemianesthesia from cerebral lesions and hysteria it may be unilaterally present, with preserved reflexes, which are lost in nuclear or trunkal disease of the pneumogastric. Hysterical adductor paralysis with aphonia commonly presents a loss of sensation at the laryngeal inlet.

Laryngeal spasm is due to irritation of the recurrent laryngeal nerve or to reflex causes mainly arising in the pneumogastric periphery and acting through this branch. With the reflex action that protects the larynx from the entrance of foreign bodies we are familiar. This may be intensified by local irritation, as in catarrhal laryngitis, especially in children, giving rise to croupy cough and attacks of croup at night. The laryngismus stridulus of rickets, or tetany, or enlarged thymus, or in goitrous, pubescent girls, is due to adductor spasm. It may be dependent upon a long uvula, enlargement of the pharyngeal tonsil, or nasal conditions sufficient to provoke the reflex. Indigestion, especially in children, and more particularly in ill-nourished children, is a common source of reflex laryngeal spasm. It may, in adults, be the result of injuries to the nerve. It not uncommonly is the neurotic equivalent of asthma or migraine, with which it may alternate. The epileptic cry is due to a laryngeal and thoracic spasm. There is a variety that is sometimes called laryngeal epilepsy. It may be an element in hysterical convulsions, or the only representative of such attacks.

A partial laryngeal spasm in severe stammering sometimes induces an explosive utterance attended by evident respiratory difficulty. From overuse of the voice, especially by bad methods, a spasmodic neurosis of the larynx similar to a writer's cramp may be set up. Speech at first is fairly uttered, but the unbalanced and spasmodic action of the vocal apparatus promptly manifests itself by loss of modulation and by explosive enunciation. Laryngeal spasm is marked by a sudden onset and often comes on during sleep. There is every evidence of dyspnea, and the marked stridor, both on inspiration and expiration, serves to distinguish it from abductor palsy of the larynx. The attack is very short, lasting but a few seconds at most.

A number of spasmodic *nervous coughs* are described, such as the barking, explosive, incessant cough of hysteria, the metallic ovarian cough of young girls, and the barking cough of pubescent and masturbating boys. In all of these conditions there is a strong neurosal element that must be given first importance in etiology and treatment.

Pulmonary Branches.—The pulmonary branches of the pneumogastric with branches from the sympathetic ganglia accompany the bronchi into the lungs. It is probable that the vagus supplies motor filaments to the bronchial muscles of unstriped fiber. The nutrition of the lungs also seems to be under their control, though the vasomotor supply comes through the sympathetic. It has been noted in animals that death, after division of the vagus, is due to pneumonia. The principal pneumogastric pulmonary derangements are bronchial asthma and protracted hiccup, which in some rare cases are interchangeable.

Bronchial or Spasmodic Asthma.—The early contention of Trousseau, Williams, and others, that there occurs a spasm of the bronchioles in asthma, is confirmed by Bert and proven by Biermer. It is accompanied by turgescence of the mucosa and a characteristic exudate of mucin in the form of spirals, which often contain polygonal crystals, by Leyden supposed to be causal of the attack. There can be no doubt that this neurosis is common in some families by direct inheritance for generations. In rare cases in the same patient it has alternated with migraine or attacks of hiccup, and even with epilepsy. In other instances, again, in neurotic stock it has taken the place of graver neuroses and of the psychoses in members of the same or succeeding generations. While it may originate apparently without any cause extraneous to the individual, in many cases every attack can be traced to certain irritant factors, such as the inhalation of dust, the pollen of certain plants, or a certain odor. It has been shown by Hack and confirmed by many others that nasal conditions may incite and prolong the attacks, which do not appear when the source of nasal reflex irritation is removed. In the same way intestinal and genital disturbances may, in individual cases, be the starting-point of the attacks. Spasmodic asthma is also associated with defective renal activity and the various acute and chronic uremias. Its relation to gout, rheumatism, and plumbism is not less clear. In every instance a potential state must be present, and these peripheral or endogenous excitements merely fire the train resulting in the nervous explosion of the asthmatic attack.

Spasmodic asthma, except in the form of hay asthma, rarely appears before adult age. It is more frequent in men than in women, a fact that is to be explained by their greater exposure to the inclemencies of the weather. There is no doubt that laryngeal and bronchial irritation from such cause may be provocative of the asthmatic attack. In long-standing cases the secondary pulmonary emphysema and chronic bronchitis constantly keep the spasmodic features within striking distance.

Symptoms.—Asthmatic attacks come on, as a rule, with considerable suddenness, and are marked by intervals of practically complete relief. Except in the hay-asthma varieties, the patient most frequently is awakened from sleep by distressed breathing that rapidly grows worse. Inspiration and expiration are both difficult, and expiration is greatly lengthened. As the dyspnea increases and persists the face is suffused and the lips become bluish. The patient is covered with perspiration and evidences his distress only too plainly by the laboring chest, the anxious and drawn face, and the noisy, blowing, wheezy respiration. The thorax is rounded, the diaphragm depressed, and its excursions diminished; the muscles at the neck are in strong relief in the attempt to increase the respiratory action. The patient calls for air. He often props himself up in bed or on chairs and other objects to raise and fix the shoulders, thereby increasing the action of the adventitious respiratory muscles. At last, when he seems unable to endure longer, the spasmodic breathing lessens, relief is experienced, and he may fall into the sound sleep of exhaustion, perhaps to be again awakened after a few hours by a repetition of the attack. The first attacks are usually mild, and only attain the indicated intensity after a number of asthmatic bouts. In the advanced and ancient cases a small degree of spasm may be continually present, manifesting itself upon the slightest exertion or exposure. During the attacks the physical signs are very slight. Roughened bronchial breathing and moist râles are noted. At first hard coughing brings up a little mucus, but toward the end of the attack a considerable quantity is frequently ejected with apparent ease and relief.

Treatment.—In the management of asthma the neurotic condition should ever be kept in mind. General measures to build up the tone and stability of the nervous system are essential. Outdoor life and moderate exercises, hydrotherapy, tonics, correct habits, and hygienic measures are indicated. The inciting element must be most carefully and persistently sought. If this can be recognized early, its removal gives hope of complete immunity from the recurring attacks that in so many cases make life wretched. It is probable that every repetition of the asthmatic storm reduces the power of resistance and serves to establish an increasing asthmatic habit. When this has been formed, and bronchitis with emphysema induced, a cure can not reasonably be expected. Regarding drugs, the iodid of potassium has a deserved reputation. It is particularly indicated in the chronic and uremic forms of asthma. Strychnin in large and increasing doses gives occasional aid. It may be given in doses increasing to $\frac{1}{10}$ of a grain three times a day, if well tolerated. The correction of peripheral states in the nose, intestines, kidneys, genitals, etc., has been sufficiently urged. Climatic changes are of importance, but one can never predict the result of this measure. In every case it is experimental. One patient will have complete immunity in a locality that is unbearable to another apparently similar case. In hay asthma a patient may secure relief one year at some resort and the following season find his journey futile.

The attack can often be cut short by the use of any one of a number of sedatives. Inhalations of nitrite of amyl or chloroform may give almost instant relief. Fumigations with niter and stramonium or some similar solanaceous plant are much in favor. Cigarettes and pastilles of such materials are prepared by the trade. The smoke must be deeply and freely inhaled. A dose of spirit of chloroform or of sulphuric ether is often productive of temporary benefit. Inhalation of steam charged with camphor is a ready and sometimes efficient measure. This is furnished by dropping a dram of any camphor preparation into a pint of boiling water in a small pitcher, over which the patient holds his face.

Hiccup is usually referred to the phrenic nerve, the diaphragmatic action being considered its most important feature. It appears, however, to be a respiratory difficulty, and is undoubtedly associated with the respiratory centers. Not only does the diaphragm act spasmodically, but there is a general thoracic inspiratory movement and a laryngeal fixation or spasm, giving rise to the peculiar inspiratory sound with which all are familiar. In some cases there are protracted attacks of hiccup that are neurotic equivalents for asthma or other nervous disturbance. Such attacks may also occur independently. Injury to the phrenic or to the pneumogastric, or even to the superior laryngeal nerve, has occasioned it. In hysteria it is not a rare manifestation, and may last for weeks during the waking hours. It may be dependent upon distant reflex irritation in the intestine or genito-urinary tract, or upon affections of the larynx and pharynx. In all conditions when the respiratory centers are intoxicated or depressed, as in uremia, syncope, suffocation, after hemorrhage, in cholera, etc., hiccup may appear, and is of serious significance.

The treatment of a protracted attack of hiccup is etiological. Some rebellious cases have been promptly terminated by inducing sneezing, which is the exact opposite of singultus. Sedatives of all sorts have at times seemed useful, and musk is especially recommended. Faradism to the phrenic nerve and diaphragm has served a good purpose. In the hysterical cases often nothing short of complete isolation and the most thorough management of the hysterical condition will avail.

Cardiac Branches.—The cardiac plexus is made up of accelerator branches from the sympathetic, and of the superior and inferior cardiac branches of the vagus, respectively given off from its cervical and thoracic portions. The vagus filaments are known to inhibit the heart's action, and are supposed to subserve sensation, being afferent in this function. Whether or not they contain trophic fibers for the heart is still disputed. Affections of the cardiac branches of the pneumogastric, or neuroses acting through these branches, modify their inhibitory function, or give rise to cardiac sensations, or both.

Tachycardia is undue rapidity of the heart's action. It may be temporary or permanent, and is due to the increased accelerator sympathetic influence or to decreased vagus inhibitory control, or to both acting together. Some individuals have naturally a quick heart. The term tachycardia is here limited to an acquired rapidity aside from that of exertion, that associated with elevation of body-heat, hemorrhage and weak heart, convalescence from acute illness, etc. It may be temporary or permanent, but usually occurs in attacks or paroxysms in which the patient feels distressed and anxious, often presents flushings and other vasomotor disturbance, and the pulse may be found beating at 120 or even attain a rate of 300 or more a minute. Of this the patient may be unconscious, in which respect it differs from palpitation, an essentially subjective sensation.. The attack often terminates rather suddenly, and may be followed by free sweating, copious urination, or even by diarrhea. Nothnagel gives the following distinctions between accelerator irritation and vagus paralysis: Great increase in frequency of pulse, with weak heart-sounds and other disturbances in the pneumogastric area, as aphonia, hoarseness, gastralgia, or cardiac pain, refer to the vagus. Strong heart-sounds and impulse, full peripheral vessels, and vasomotor storms indicate accelerator disturbance. Whittaker[1] says the increase to 120 beats implies irritation of the sympathetic; from 120 to 180 beats, paralysis of the vagus; above 180, the combined effect of both causes.

Tachycardia may be due (1) to disease of the heart and blood-vessels, (2) to injury of the vagus trunk or nucleus, (3) to toxic causes,—alcohol, nicotin, coffee, and atropin,—(4) to a reflex from any viscus, especially those in the pneumogastric field, and (5) to many neuroses, as Graves's disease, hysteria, and neurasthenia. The prognosis and treatment necessarily depend on the causation. The purely neurotic forms are difficult to manage, though not of serious import so far as life is concerned.

Cardiac palpitation is sometimes a purely nervous condition, a vagus neurosis. Hysterics and neurasthenics are often much troubled by this rapid heart-action, of which they are perfectly aware. It may come on independently of any assignable cause, as during moments of rest, or even during sleep, though here the influence of some distressing dream can not be excluded. It lacks the anxiety and often associated heart-pain of tachycardia and angina pectoris, but is often attended by tinnitus, vertigo, and a feeling of faintness. All source of cardiac disturbance must be excluded before admitting this form of vagus disturbance.

Unless it is merely symptomatic, the etiology is obscure and its treatment difficult. Sedatives and narcotics, with local applications of heat or cold over the precordium and the administration of diffusible stimulants, may be employed to arrest the attack.

Bradycardia is the opposite of tachycardia. A slow pulse is apparently normal to many individuals and is not associated with any distress or difficulty. Such instances, in which the heart-beats are twenty, thirty, or forty a minute, are on record. The term is here limited to an acquired slow beat that may be permanent, temporary, or paroxysmal. It is sometimes noted in cervical myelitis or injuries to the cord in this region. Cerebral conditions marked by pressure, as hemorrhage, hydrocephalus, and tumor, reduce the pulse-rate, and it is often slow in meningitis. The actions of narcotics, biliary poisoning, and uremia only need to be mentioned. Many infective diseases, such as rheumatism, puerperal fever, typhoid, and diphtheria, may produce bradycardia of a persisting sort, or may be followed by it. They probably, at times, set up a vagus neuritis. Organic processes acting on the pneumogastric cardiac inhibitory center, or on its cardiac fibers, may retard the heart by the irritant stimulation of the inhibitory function. Disease of the heart-muscle itself, as in fatty heart and coronary sclerosis, may produce bradycardia, and seems to act by irritation of the terminal filaments of the vagus. The nuclear variety is often associated with arteriosclerotic changes in the medulla.

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[1] "Twentieth Century Practice," vol. iv.

Bradycardia is usually attended by syncopic disturbance, which has not rarely been mistaken for epilepsy. The paroxysmal form is often marked by anxiety, unrest, and distress in the precordial region. Cardiac angina and nausea, or vomiting, may ensue. Convulsions are sometimes noted. When the vagus trunk is affected, other motor symptoms may indicate it, such as aphonia, hoarseness, stridor, and choking. The diagnosis of bradycardia is not difficult, but the heart-beat should never be determined by the radial pulse. The treatment and prognosis depend on the causal condition.

Angina pectoris is an affection of the vagus marked by paroxysms of agonizing pain in the region of the heart, which radiates usually to the left shoulder and arm, and is attended by a sensation of impending death. By some it is called true angina pectoris when dependent, as is usually the case, upon organic heart disease, in distinction from false or pseudo-angina, in which no anatomical changes are discoverable. There is a strong probability that the sympathetic fibers participate in the storm, but the essential element is the vagus neuralgia. The attacks are sometimes induced by exertion or emotion, but may rouse the patient from a sound sleep. Rarely occurring in children, it is much more common in male adults and in the latter half of life, when the arterial changes and cardiac myopathies are commonly found. It may occur independently of such organic changes in neurotic individuals, and is encountered in hysteria, subsiding with that neurosis or suddenly ceasing upon the appearance of other hysterical manifestations.

The pain varies in character and severity. Its usual radiation to the brachial plexus may be replaced by epigastric, ilioscrotal, vesical, and even sciatic pain, ordinarily on the left side. The feeling of impending death, however, is an essential symptom. The pulse may be unchanged even in the presence of the most excruciating pain and the most frightful anxiety, or, rarely, the heart may present tachycardia or bradycardia. The patient is often bathed in perspiration, and in rare cases other pneumogastric conditions, as asthma, laryngeal spasm, or esophagismus, may attend the anginal attack. The attack lasts from a few minutes to an hour, and usually subsides as suddenly as it commenced. The patient feels considerably shaken up and rather languid for a time, but in the intervals of the attacks may be completely free from all distress aside from that attributable to organic cardiac effects and the apprehension of another attack.

The prognosis depends on the condition of the heart. If it is not organically diseased, the angina does not end fatally, and even cases of organic heart disease seldom die in anginal attacks. The treatment of the attack is antispasmodic. Amyl nitrite and trinitrin hold the first place; chloroform by inhalation, the application of heat or cold to the precordium, whisky, and other similar measures are of some value. In the intervals treatment is directed to the condition of the heart or to the neurosis, or to both.

Gastric Branches.—The pneumogastric supplies motor branches to the stomach, but only in part innervates the muscular coat of that viscus. Certain gastric movements, such as sobbing and vomiting, undoubtedly depend upon its motor filaments. Vomiting is reflexly produced through its gastric sensory fibers, as well as directly by intracranial disease or irritation of its trunk. Vomiting due to intracranial disease, or that provoked by irritating the vagus trunk and sometimes that due to irritation of its stomach branches by organic disease, is peculiarly rapid, projectile, and, as a rule, unattended by nausea. The vagus probably conveys the sensations of hunger, at least they have been completely destroyed by bilateral lesions of these nerves. Ravenous appetite for food and the peculiar subjective gastric sensations of dyspepsia, organic disease of the stomach, and some neurotic and psychical states are properly attributable to the central or peripheral conditions of the pneumogastric nerve.

Gastralgia, or Gastrodynia.—Aside from the stomach-pain of organic gastric disease, fermentation, and dyspepsia, there is a neurosis of the stomach marked by sudden pain in this viscus, to which the name nervous gastralgia is given by Ewald. It is not attended by the symptoms of disturbed digestion, and the stomach contents at such times, as well as between the attacks, show nothing chemically irregular. It may alternate with other neuralgias or with migraine, or, rarely, it is associated with an attack of the latter. It is occasionally presented by hysterics, particularly if their attention is centered on the stomach. The gastralgic attack usually comes on promptly and reaches its highest intensity almost at once. The pain is an agonizing, boring, cutting, burning one, and may be localized, diffuse, or in a girdle. It may radiate along the loins or spine. The patients relax the abdominal walls and make deep pressure over the stomach with some relief, though there may be much superficial sensitiveness. Persistent sensitive spots are often found over the lower dorsal vertebræ, between the ribs, and by deep pressure over the abdominal plexuses of the sympathetic. The face is pale, distorted with pain, and covered with sweat. The temperature is not modified. The attacks may be of only a few minutes' duration or may last for hours, and usually terminate rather abruptly, often with the vomiting of mucus or unchanged food. Food is then often actively craved and taken without distress.

These rather rare attacks may very easily be mistaken for gall-stone colic, acute indigestion, gastritis, gastric ulcer, and a host of other abdomprominence in the muscles it supplies. By the sternomastoid the face is carried forward, turned toward the opposite side, and tilted upward. The trapezius draws the head backward and to the same side, lifts the shoulder, and shortens the distance between the occiput and acromion. At the same time the scapula is drawn nearer the vertebral spines as well as upward. Both sternals acting together bring the head forward and downward. Both trapezii carry the head backward and the face upward. Acting alternately they turn the head from side to side with slight rotation. The spasm may be clonic or tonic, may involve one or both sides, and may be limited to the sternals or the trapezii. A tendency to spread to other muscles of allied function is often observed. As a matter of fact, in most cases of wryneck more muscles are involved than those supplied by the accessory nerves. Thus the splenius and the small rotators are frequently associated in the spasm, just as they are related functionally. The large muscles named are, however, the ones most at fault and the ones that impress a given case with its distinctive movement or attitude. The trapezius is less often clonically involved than the sternomastoid, but is as frequently affected by tonic spasms. In some cases clonic torticollis is a localized tic, or it may be a portion of a more widely distributed tic such as that arising in the face and gradually involving the neck, shoulder, and arm. In a mild form it furnishes the habit spasm that follows chorea sometimes or is picked up by pubescent youths. We may also have a mental torticollis as described by Bompaire,[1] in which the patient can not voluntarily restore the head to its natural position, but turns it readily in all directions if allowed to press it against any object, or to apply an insignificant amount of manual assistance. The spasm is here the result of a mental obsession. The nodding spasm of hysteria arises in the same way, and in both the disorder must be referred to the cortex. In this connection the subject of tics in Part VII should be read. Cerebral tumors, meningitis, focal softenings, and disease of the medulla may underlie the spasm. Cervical spondylitis and meningitis usually produce tonic spasms. The action of cold, cervical adenitis, and violent wrenching of the neck may peripherally excite them. Torticollis has been reflexly produced by intestinal worms.

Treatment is often highly unsatisfactory. The variety due to cold usually subsides spontaneously or may be benefited by local applications and antirheumatics. The hysterical form may suddenly cease. The cases dependent upon organic lesions are often beyond reach. Sedatives like → cannabis indica, opium, and hyoscin only give temporary relief and often upset the stomach or, unfortunately, lead to their habitual use. When the spasm is severe, and especially if confined to one side, wide resection of the spinal accessory nerve before its entrance into the sternomastoid is advised, other means having failed. A tic or habit is thus likely to be benefited for a time, but is also likely to reappear in some adjoining area. Stretching the nerve is almost sure to be followed by a relapse of the tic as the nerve recovers, as is the case also if the divided nerve unites.

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[1] "Thèse de Paris."

The upper spinal nerves to the small rotators must in some cases also be divided to completely quiet the spasm.

Children sometimes present a permanent wryneck of an entirely different character. Owing to traction on the head and twisting of the neck in labor, or even in unaided labor, the sternomastoid may be injured, and subsequent contracture shortens it. The result is a firm, fibrous band that holds the face to the opposite side. Inflammatory injury to the muscle in adults may cause the same thing, and in both cases it can be remedied only by thorough division of the shortened tissues.

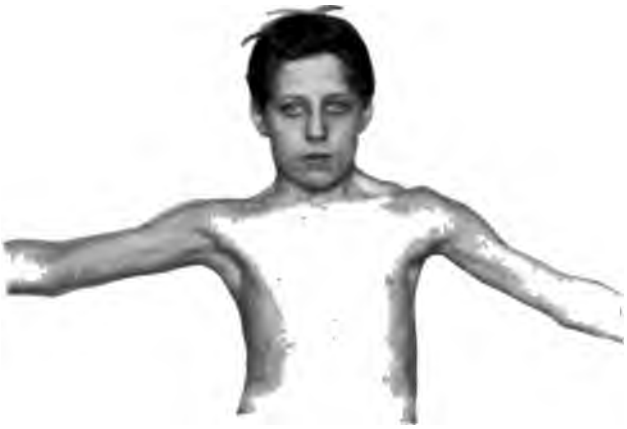
Fig. 61.—Difficulty in raising arm after division of left spinal accessory.

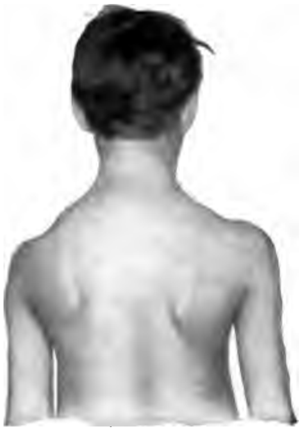
Fig. 62. — Drooping shoulder, lengthening of neck, and rotation of scapula after division of left spinal accessory.

Accessory Paralysis.—Cortical disease very exceptionally results in complete and permanent loss of power in the spinal accessory area. Like those of other bilateral movements, these muscles seem fully represented in both hemispheres. As a part of nuclear disease, as in progressive muscular atrophy, the spinal centers are often involved, with a corresponding loss of power and nutrition. By meningitis the trunks of one or both nerves may be implicated at the foramen magnum. The hypoglossal is then likely to suffer with them, and the true accessory fibers are usually also affected. Outside the skull the spinal portion of the accessory is sometimes involved in wounds, operations, deep-seated tumors, vertebral caries, cervical adenopathy, and by neuritis. The unilateral symptoms are loss of power and wasting in the related muscles. This involves the sternomastoid almost entirely and the trapezius only in its upper portion. The head is not so readily and strongly turned to the opposite side, the shoulder droops slightly, and the extension power of the arm is lessened. The scapula moves outward, especially its upper inner angle, and stands out from the ribs. The curved line from mastoid to acromion becomes depressed and even angular upon efforts at deep inspiration or in extending both arms against resistance. Subsequent contracture in the unopposed muscles of the sound side may turn the face to the primarily affected side.

In the bilateral form, usually due to meningitis or vertebral caries, the head is held insecurely and readily falls forward or backward as the trapezius or sternals are most affected. Injury to the spinal accessory in the posterior triangles of the neck after it has passed through the sternomastoid only affects the trapezius. The treatment is that of the causal condition in the given case. In neuritis electrical stimulation is indicated, and nerve-suture would be required in the cases where the nerve had been divided.

CHAPTER XI.

DISEASES OF THE HYPOGLOSSAL NERVE.

Anatomical Considerations.—The twelfth cranial nerve is a purely motor nerve for the muscles of the tongue and subserves their nutrition. Its cortical centers adjoin those for the lips in the lower portion of the ascending frontal gyre. Its lower neuron arises in the hypoglossal nucleus under the floor of the fourth ventricle. This nucleus is close to the median line, and is made up of large cells analogous to those in the anterior spinal horn of gray matter. The nerve-trunk arises by a number of roots in the groove outside of the olivary body, and, passing forward, leaves the skull by the anterior condyloid foramen of the occipital bone. It has a connection with the pneumogastric, which it accompanies a short distance, and receives a branch from the upper spinal nerves. This branch eventually innervates the depressors of the hyoid bone, and is not affected by injury to the hypoglossal nucleus. The hypoglossus may be injured or diseased in any part of its course from the cortex to the peripheral branches, and manifests such injury by spasm, paresis, and paralysis, as the causal state is irritant, inhibitive, or destructive. In addition, injury at or below the nucleus entails atrophy of the muscular fibers of the tongue. The mucous portion of the tongue is supplied by the trifacial.

Hypoglossal Spasm.—The tongue is usually involved in epileptic convulsions, and is frequently bitten in consequence of having been thrust between the teeth by the action of the genioglossus muscles and lacerated by the spasmodic action of the masticators. This may occur in very slight attacks, when no convulsive action in the extremities is observed. At an early stage of hysterical convulsions the tongue is usually protruded violently, but is only bitten in extremely rare cases. This lingual spasm may comprise the entire motor disturbance in some hysterical cases. In chorea the tongue is usually involved in the muscular twitchings that mark the disease, and is often the seat of vigorous choreic movement, in which it may be protruded and even slightly bitten. The speech difficulties of this disease are due in part to the lingual spasm. From forced overuse, as in public speakers, it is sometimes the seat of a neurotic manifestation similar to writers' cramp, a condition termed aphthongia. Slight spasm also occurs in stammering and in stuttering. Lingual spasm has been reflexly dependent upon decayed teeth and cured by their removal.

Cortical irritation has, in rare instances, given rise to lingual spasms that practically constitute Jacksonian epileptic attacks. The lips and face usually participate in the spasm, as the contiguity of their centers would lead one to expect. In some of these cases the initial convulsion has been a generalized fit; in others such attacks have followed. It is conceivable that nuclear irritation might give rise to similar manifestations, but post-mortem data for this belief are lacking. The treatment would be that of the general condition underlying the local manifestation.

Hypoglossal Paralysis.—The hypoglossus nerve may be paralyzed by injury at any point in its course. In rare cases a limited cortical lesion has produced paralysis of the opposite side of the tongue. More wide-spread cortical lesions and lesions in the supranuclear motor tract usually produce a one-sided paresis or paralysis of the tongue, which, on protrusion, deviates toward the paralyzed side,—that is, away from the lesion. This is due to the action of the unaffected and properly acting genioglossus of the sound side. Nuclear disease usually produces bilateral palsy of the tongue, which lies motionless in the floor of the mouth, and, after a few days, rapidly wastes. A few cases of one-sided nuclear disease are recorded. In bulbar palsy, syringomyelia, and tabes it is thus affected in exceptional cases. Other cranial-nerve nuclei usually suffer at the same time, and the resulting symptoms enable one to localize the disease. The fibrillar twitchings in the tremulous tongue of paretic dementia and some of the stammering of this malady are due to the nuclear and cortical invasion of the disease. The root of the nerve in its passage through the medulla is sometimes damaged by a local lesion, which of necessity interferes with the pyramidal tracts for the opposite side of the body and produces a crossed paralysis of the tongue and limbs. The tongue, in such an instance, would deviate from the paralyzed side of the body and toward the side of the lesion. Such a crossed palsy, due to disease of the olivary body, has been reported.[1] Basilar processes, like meningitis and fractures, or bone disease involving the condyloid foramen, may injure the nerve in its intracranial course. Outside of the skull it is vulnerable to penetrating wounds, or may be implicated in deep abscesses, as from caries of the upper vertebræ. Here its neighbors, the spinal accessory and the pneumogastric, are likely to be involved at the same time. It may be affected by a neuritis as in a case reported by Panski,[2] who was able from the literature to collect 40 cases of isolated hypoglossus paralysis due to peripheral disease or injury.

In unilateral hypoglossal paralysis due to damage of the nerve at or below the nucleus, the paralyzed side of the tongue shows a marked loss of volume. This does not follow supranuclear lesions. The mucous covering, on the other hand, not being deprived of its trophic supply, is thrown into marked and apparently excessive folds. Taste and sensation are not impaired. Mastication on the paralyzed side of the tongue is not well performed, as the patient finds difficulty in placing and maintaining the food between the teeth. When protruded, the tongue curves sharply to the wasted side, but within the mouth motions toward the palsied side are wanting. In the bilateral form speech is much affected, as nearly all consonant sounds depend in some degree upon the position of the tongue. Swallowing is also difficult, as the bolus or fluid is not readily carried backward into the pharynx and mastication is greatly impeded.

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[1] Gonkovsky, "Nouvelle Iconographie de la Salpêtrière," No. 3, 1895.

[2] "Neurolog. Centralbl.," Aug., 1903.

Treatment is directed to the causal condition, aside from which it is practically hopeless.

CHAPTER XII.

MULTIPLE PARALYSES OF CRANIAL NERVES.

In discussing the cranial nerves separately, frequent reference has been made to their association in diseased processes. Sometimes the limitation of this association is of diagnostic importance, as when the loss of hearing with facial palsy locates a lesion of the seventh and eighth nerves in their parallel intracranial course or within the internal meatus auditorius. In meningitis and other basilar processes a number of cranial nerves are usually implicated at once, and such multiple paralyses of cranial nerves become symptomatically highly important. The close juxtaposition of the cranial nuclei has been repeatedly pointed out as giving rise to associated palsies in bulbar disease, such as tumor, hemorrhage, and limited basilar thrombosis. In their cortical fields and their supranuclear motor paths a number of the cranial nerves may also be injured at once by traumatism or disease. Hypoglossal palsy with hemiplegia has just been called to attention, as well as the association of hypoglossal and facial spasm due to the cortical relations of their centers. These multiple cranial palsies are principally of symptomatic interest and value, but there are groups of nuclear palsies of the cranial nerves that present well-marked clinical forms of disease. Their pathological anatomy is largely confined to the upward extension of the spinal gray that furnishes the chain of cranial-nerve nuclei reaching from the lower angle of the floor of the fourth ventricle to the posterior portion of the third. Anatomically and clinically these diseases fall into two forms: First, those of the upper group, primarily implicating the nuclei of the third, fourth, and sixth nerves (see Fig. 36), and, second, those first implicating the remaining lower cranial nuclei. As these diseases are sometimes inflammatory in character and analogous to poliomyelitis, they have come to be known respectively as polio-encephalitis superior and polio-encephalitis inferior. More commonly the process is one of degeneration. The analogy, or in some cases the identity, of the disease of the nuclei of the bulb with that of the anterior gray of the cord is now generally conceded. It is the same process arising in different localities, but affecting homologous elements and producing results similar in kind. Transition and combination cases of every variety are of record. Commencing in the upper nuclei of the ocular nerves, the lower bulbar levels may be progressively invaded, and later spinal features may be added; or, beginning in the bulbar nuclei, an upper extension may induce progressive ophthalmoplegia. In the same way that the spinal type of the disease may be acute, subacute, and chronic, we have an acute, subacute, and chronic polio-encephalitis superior and inferior, and forms combining both.

Polio-encephalitis superior chronica will be considered first, as it presents the most complete and well-defined picture. It is also called *progressive ophthalmoplegia* and *chronic nuclear ocular paralysis*. It is characterized by the gradual loss of power in the external and internal muscular apparatus of the eyeball, one set of muscles after another being gradually involved. Usually bilateral, it may be confined to one eye for a period even of years and then invade the other. As a rule progressive, it may come to a permanent standstill at any time, or advance intermittently.

Etiology.—Polio-encephalitis superior has in so many cases followed infections and toxic states that it has with reason been attributed in some cases to pneumonia, grip, diphtheria, and syphilis; to lead, sulphid of carbon, carbonic-oxid gas, sulphuric acid, and alcohol. These toxic agents, however, are more common in the causation of the acute variety, which may terminate by becoming chronic. It may be secondary to or a part of tabes dorsalis, and the characteristic Argyll-Robertson pupil of that disease is a part of it. It is similarly related to posterolateral spinal sclerosis, general paresis, and insular sclerosis. It may be an upward extension of identical disease in the cord or bulb. It is more frequent in men than in women, and while it may appear at any age, it is principally a disease of adult life. In some cases there seems to be a teratological defect, congenitally manifest or appearing during the active periods of growth.

Pathological Anatomy.—The lesions are variable in extent. They involve the upper cranial-nerve nuclei on one or both sides, including those of the third, fourth, and sixth nerves, wholly or in part. When partial, the iridociliary nucleus of the third and its adjoining nucleus for the elevator of the lid most frequently escape or are only partly destroyed. The corresponding nerve-trunks and muscles are degenerated and atrophied. In addition are encountered cerebral, bulbar, and spinal conditions, of which polio-encephalitis superior sometimes is but a part. Microscopically, especially in the early stage, some hyperemia is found about the nuclei. Later the cells of these nuclei lose their prolongations, are diminished in size, present vacuoles and pigmentary degeneration. Some completely disappear, and in cases of long standing but a few atrophied remnants remain. Slight capillary hemorrhages or their traces can at times be determined. Some leukocytal infiltration occurs about the vessels or in the foci of the disease, and minute sclerotic patches are found. Rarely the lining of the Sylvian aqueduct is thickened.

Symptoms.—In the fully-developed cases the facies, classically described by Hutchinson, and recognized by Brunner, von Graefe, Charcot, and others, is most striking. The drooping lids partly covering the eyes cause the patient to hold the head back and strain with the frontalis to overcome the partial ptosis. The brow is thrown into deep transverse wrinkles. The immobile eyes and inactive pupils are like those of glass in a mask of wax. They usually deviate outward, as the sixth nucleus is often spared at first; but any squint may in various cases be present or the visual axes may conform. The ptosis is usually less marked after a night's rest; and if the ocular muscles still retain a slight degree of activity, this, too, is best in the morning. Diplopia is rarely mentioned by these patients, which Blanc attributes to the slowness with which the deviation develops, thereby allowing the brain to neglect one image. In partial cases the ocular conditions may be unilateral or they may be only functionally distributed and bilateral or unilateral. Thus the iris may react for light or accommodation, or both. The abducens may at first escape or first be involved, and innumerable combinations and varieties have been encountered as one or many nuclei were partially or completely affected.

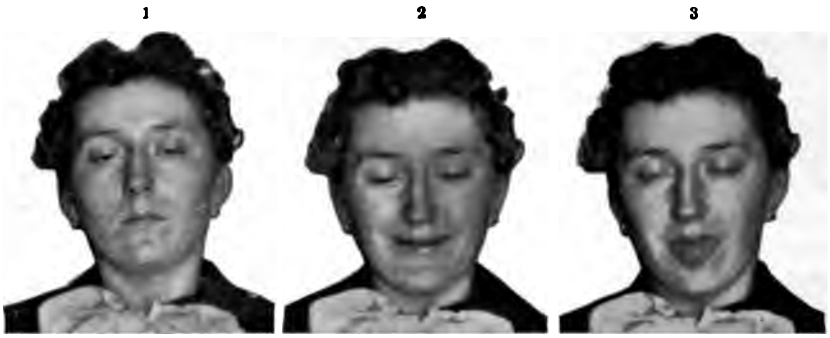

Fig. 63.—Polio-encephalitis superior and inferior. 1, Myopathic facies; mask-like, expressionless face, drooping eyelids, etc.; 2, forced attempt to smile vigorously; 3, forced attempt to close eyes firmly and to protrude tongue vigorously: it passed the lips with difficulty; the eyeballs were hardly covered.

The general health of the patient may leave little to desire or he may present the somatic conditions and functional disturbances that belong to the associated diseases already mentioned. In some instances involvement of the fifth nerve, probably through its descending root, has given rise to paresthesia or anesthesia in the face.

Course.—The evolution of the disease is one of its most distinctive features. Commencing with a ptosis or a squint, the other features of the disease are gradually added during, perhaps, several years. Stationary periods of long duration—twenty-five years in a case of Strümpell's—may intervene, and finally the disease takes up its progressive course, perhaps without appreciable cause. Extensions of the process to the bulbar region may occur, and we have bulbar palsy or polio-encephalitis inferior added and the prognosis for life becomes much darkened. The lesions may not stop at the bulb, but may invade the cord, producing a progressive spinal muscular atrophy of any one of the various types of that disease.

The diagnosis depends largely upon the evolution of the disease and its progressive course. When thoroughly developed its remarkable facies is not easily mistaken. The acute form, and hemorrhage, inflammation, and softening, give a history of rapid onset and promptly reach their maximum stage. In multiple neuritis, especially the alcoholic and diphtheric varieties, confusion is likely to arise, but we may generally be guided by the condition of the extremities and the wide diffusion of the disease. Even then we can not absolutely exclude the cranial nuclei, which may become the seat of a chronic atrophic process. An orbital tumor may give rise to a partial and increasing ophthalmoplegia, but pressure symptoms, exophthalmos, and optic atrophy will finally distinguish it. Basilar processes and bulbar growths, aside from the distinctive symptoms to which they give rise, usually implicate so many nerves and the pyramidal tracts that they can be easily separated.

Treatment turns practically upon the presence or suspicion of syphilis, which should be vigorously managed. Other toxic possibilities, such as lead-poisoning, will require appropriate intervention. Strychnin does temporary good sometimes. Electricity is futile, as it can not be applied to the faulty muscles. In the majority of cases the physician is helpless.

Acute and Subacute Polio-encephalitis Superior.—The acute form is very rare. Like its congener, acute poliomyelitis, it may be purely an infectious disease, and has been noted in epidemics of the spinal disease. It may follow many acute infections, as mentioned in the etiology of the chronic form, especially diphtheria, pneumonia, and influenza. The subacute form, while in some cases attributable to the above causes, is more often due to syphilis and toxic agents, like lead and the carbon gases. The onset is sudden or very acute, and the disease may reach a fatal termination in a few days or a week, usually from implication of the bulbar centers. The nuclei in several such cases have presented no post-mortem changes that could be discovered. Brissaud [1] supposes that the infection has overwhelmed the nuclei before histological traces discoverable by our present means of investigation were developed. The similarity to Landry's paralysis in this regard is striking, and the diseases may be essentially the same, varying only in localization. In other cases well-marked inflammatory changes have been discerned. The prognosis is very grave, especially in children. Some cases come to a standstill; others develop into the chronic variety.

The treatment should be directed to securing perfect rest and the removal of any source of infection or intoxication and its eradication from the system before important changes occur.

Polio-encephalitis inferior chronica is the term employed by Wernicke to denominate the disease first described by Dumesnil, later by Duchenne, as progressive paralysis of the tongue, palate, and lips. It is also known as *labioglossolaryngeal* or *pharyngeal paralysis* (Leyden), as *progressive bulbar paralysis* (Wachsmuth), as *paralysis of the bulbar*

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[1] "Lecons," 1895.

nuclei (Kussmaul), and as *progressive bulbar palsy*. It consists in a progressive paralysis of the lips, tongue, pharynx, and larynx, with wasting of their muscles, and usually terminates in death from pneumogastric palsy. The disease process is limited to the nuclei and lower neurons of the seventh, ninth, tenth, eleventh, twelfth, and the motor portion of the fifth cranial pairs. It is identically the same in character with progressive spinal muscular atrophy, with which it is often associated.

Etiology.—Males and females are about equally susceptible to this disease. Though it has rarely been observed in childhood and may be a congenital or teratological deficiency, it usually appears after the age of thirty. Occasionally, and then ordinarily as a part of a more extensive spinal atrophy, or in association with superior polio-encephalitis, it has appeared in successive generations or in more than one member of the same family. These family cases usually develop in early life. It has been attributed to exposure to cold; to the overuse of the mouth-muscles, as in players of wind-instruments and glass-blowers; to syphilis, to Bright's disease, and other wasting maladies. It may be a part of amyotrophic lateral sclerosis. It has appeared in tabes dorsalis, insular sclerosis, syringomyelia, and followed descending degeneration of the pyramidal tracts after cerebral lesions. Knowledge of the rôle of toxemia in these chronic disturbances is widening. Progressive bulbar palsy has been known to follow lead intoxication and diphtheric poisoning. Often the causation is entirely obscure, but advances in the field of auto-intoxication may throw light on this darkness.

Morbid Anatomy.—The lesion is limited to the nuclear cells in the lower half of the bulb. It is practically symmetrical. The hypoglossal nucleus is most severely affected, as a rule. The nuclei of the true spinal accessory, the facial, the motor portion of the trifacial, and the pneumogastric are invaded with decreasing intensity and frequency. A degenerative process is found in the nerve-trunks whose nuclei are affected and their muscular terminations waste. The muscle-fibers themselves show corresponding degeneration and atrophic conditions. The minute anatomy is the same as that in progressive ophthalmoplegia or that of progressive spinal muscular atrophy, to which the reader, bearing in mind the special location of this disease, is referred. The organic changes of associated atrophies and scleroses or of primary conditions, such as tabes, syringomyelia, insular sclerosis, and descending cerebral degenerations are at times encountered.

Symptoms.—The symptoms begin insidiously and progress slowly. The *tongue* is usually the first affected. This is manifest in a thickened pronunciation, particularly of the letters which require definite lingual movements. The linguodentals and linguopalatals (see table, p. 66) and the vowel "e" and later on the explosive labial sounds are lost. Finally speech is reduced to unmodified laryngeal noises that are quite unintelligible. When the disease is only slightly developed, by an effort the patient can often enunciate clearly and the embarrassment may be noticed only in ordinary inattentive conversation. The tongue also progressively loses its muscular strength and range of motion until it lies a flabby, inert, rugose, atrophic mass on the floor of the mouth. It loses its function of maintaining the food between the teeth during mastication and of carrying the bolus backward and thrusting it into the pharynx in efforts at swallowing. It can no longer be protruded, turned to either side, rolled up, or hollowed into a gutter. In most of the cases it notably wastes, but as the atrophy is confined to the muscular portion of the tongue, the dermal covering appears too large and may even suggest epidermal hypertrophy.

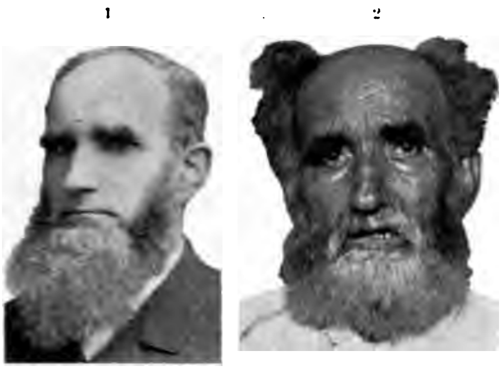

Fig. 64.—Case of bulbar palsy. 1, Photograph taken shortly before first symptoms were noted; 2, photograph taken four years later, a few weeks before death.

The *lips* are affected shortly after the tongue, and this adds to the speech difficulty by rendering the pronunciation of the vowels "o" and "u" uncertain or impossible. The labial consonants are lost as above indicated, and little besides the sibilant "s" and the open vowel "a" remain. The orbicularis oris is usually the first labial muscle invaded, but all of the labial group are eventually paralyzed. Their nuclear association with the hypoglossus and their functional relationship will be recalled. At first whistling and blowing efforts are weakened, but finally the mouth hangs loosely, the lower lip drooping away from the teeth, and all voluntary movements are lost. This, with the action and eventual contracture of the zygomatics, serves to greatly accentuate the nasolabial furrows and imparts a demented appearance to the lower portion of the face.

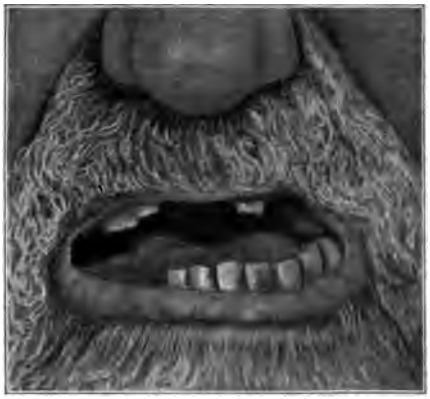

Fig. 65.—Mouth in bulbar palsy. Voluntary maximum opening and attempt to project the tongue, which lies inert and shrunken in the floor of the mouth.

Out of the drooping, open mouth saliva drules, requiring the constant use of a napkin or handkerchief. The quantity is sometimes enormous and always appears greater than normal. When the *masseters* and *pterygoids* are involved, which is usually at a late stage, but may be an initial condition, mastication is feeble or impossible. Finally, their complete paralysis allows the mandible to hang loosely, increases the opening of the drooping mouth and the salivary overflow. The jaw-jerk is abolished except in those cases where spastic symptoms elsewhere point to the association of the pyramidal tracts in the lesion. It is then increased. The lips usually show marked atrophy and are sensibly thinned. This is sometimes obscured by the fatty deposit, but in the final stages is practically a constant condition.

The *palate* follows the lips in order of involvement in a majority of cases. Its loss of muscular tone is manifested by the nasal voice tones, which also adds to the lack of clearness in the pronunciation of the linguo-palatals, and it turns " p " and " b " into the nasal resonant " m." When the palate is fully palsied it hangs loosely in the pharynx without reflex action or voluntary movement. As it can no longer shut off the nasal spaces, fluids often regurgitate through the nose, and even food masses may be forced into the nasal fossæ.

When the *pharynx* is involved, the difficulties of swallowing reach their maximum. Food is now prone to enter the respiratory tract and a violent, fatiguing, and alarming cough is often produced. The danger of pulmonary engorgement, aspiration pneumonia, and heart-failure is intensified by the pneumogastric weakness that is often present. Alimentation becomes so difficult that the esophageal tube must be used or the patient must be fed by the bowel. To these difficulties is added, sooner or later, a paralysis of the *larynx*. The adductors are usually most affected and the glottis stands wide open, serving neither the purposes of phonation nor protection to the trachea against the entrance of foreign material. In very rare cases unilateral or bilateral abductor palsy is found, and in the latter case inspiratory stridor indicates the dangerous respiratory difficulty. Both pharyngeal and laryngeal reflexes are abolished. The voice is extinguished. Failing respiratory and cardiac actions lead to a fatal termination, which may come suddenly at any period of the disease. It is often induced by aspiration pneumonia or caused by suffocation due to blocking of the respiratory passage by a mass of food.

The *pulse* is likely to become frequent, weak, and irregular. True anginal attacks are not infrequent. Syncope may occur and prove fatal. The pneumogastric involvement further shows itself in a feebleness of respiration, so that coughing and other active expiratory efforts become almost impossible, adding greatly to the danger of choking and to the general discomfort of the patient. It is somewhat remarkable that polyuria and glycosuria are seldom encountered. The controlling centers in the bulb are in close proximity to those invaded by this disease. It will be noticed that the nuclei selected are purely motor and trophic, and that the disease spreads not so much by contiguity as along lines of associated function. This is one of the distinctive habits of the disease, and serves here, as in other progressive maladies of the cerebrospinal axis, to draw earnest attention to this factor in the study of every case.

The *electrical examination* of the atrophic muscles presents considerable difficulty, excepting in the lower face and the masseters. The changes found are practically a quantitative reduction to all currents as fiber after fiber disappears. The reaction of degeneration is wanting. The reflexes are correspondingly diminished and abolished, excepting in those cases in which the disease early involves the pyramidal tracts. *Sensation* is not markedly affected, nor is the sense of taste notably disturbed in pure cases. The temperature remains uninfluenced throughout, save by intercurrent accidents.

Course.—The disease is one of insidious onset and its steadily progressive course is characteristic. In a few exceptional cases the progress of the disease presents intermissions, but remissions are practically unknown. The duration of the malady from inception to fatal termination may be roughly stated as from one to five years. Leyden reports one case of seven years' duration, but, on the other hand, intercurrent maladies and the suffocative, cardiac, and pulmonary accidents to which the disease lays the patient liable may cut life short at any moment. The increasing feebleness and malnutrition at the same time add to the gravity of the situation. As above indicated, the disease first manifests itself in the tongue and progressively invades the lips, pharynx, palate, masticators, and larynx. This is a usual sequence, but not a necessary one. Any modification of it may be presented. The encroachments of related nuclear disease at lower and higher levels give rise to different trains of symptoms, which, however, in their full development furnish very similar pictures. The course pursued in any given case can be understood by reference to the anatomical and especially to the functional relationship of the bulbar nuclei. The occurrence of a bronchitis, bronchopneumonia, angina pectoris, or of suffocative attacks is often of fatal import.

Diagnosis.—The diagnosis of a well-developed and unmixed case presents little difficulty. The course of the disease is of the first importance. The facies can hardly be mistaken. Palsy of both facial nerves gives rise to feebleness of the lips, but the upper part of the face does not escape and glossopharyngeal symptoms are lacking. Diphtheric palatal palsy may raise a doubt unless the clinical history of the infection is available. In this condition the lips and tongue escape, the onset is somewhat abrupt, and the usual course is toward recovery. Great difficulty may be presented in cases of multiple neuritis, but in them we have sensory disturbances, the reaction of degeneration, and marked symptoms in the extremities. When secondary to tabes, insular sclerosis, syringomyelia, and amyotrophic lateral sclerosis, the highly characteristic symptoms of these various diseases are prominent. As an extension process from the cord upward, or from the ocular nuclei downward, its development is preceded by the well-marked evidence of these prior states, which persist and increase during the evolution of the bulbar paralysis. The greatest diagnostic difficulty is presented by cases of the pseudobulbar paralyses.

Treatment.—In pure polio-encephalitis inferior chronica the prognosis is fatal. Curative treatment is, therefore, out of the question, but much can be done to alleviate the distressing condition of the patient and to obviate the laryngeal, pulmonary, and nutritive dangers that threaten him with suffocation, asphyxiation, pneumonia, and inanition. The hypersecretion of saliva may be checked by atropin, which also furnishes a reliable heart-stimulant. The stomach-tube and rectal alimentation are our means to avoid strangulation and to secure nutrition. Recourse to tracheotomy may be had in abductor laryngeal paralysis. Electricity is of use in exercising the muscles of the face, tongue, and gullet. The faradic current is sufficient. The large, indifferent electrode may be placed on the back of the neck, and a smaller, active electrode is then brought into contact with the lips, masseters, and tongue. By placing it over the pomum adami swallowing efforts are induced. Care must be exercised not to fatigue muscles already wasted. Energetic courses of silver, ergot, phosphorus, zinc, picrotoxin, and mercury are mentioned only to condemn them, and anything else that pulls down the waning strength of the patient must be avoided. Tonics, rest, and strengthening measures are of value.

Acute bulbar palsy, or *acute bulbar myelitis*, is due to the same infections that set up acute myelitis. It may furnish the terminal stage of the chronic form, or it may result from an upward extension of a cord-lesion. The symptoms with which we are familiar in the chronic variety are rapidly evolved and often associated with febrile disturbances, headache, and somnolence. As the disease gains the pneumogastric nuclei, death becomes imminent and results through respiratory failure.

Combined Forms of Polio-encephalitis.—The various combinations of chronic superior and inferior polio-encephalitis by extension have been alluded to in describing them separately. In some very rare cases the nuclear invasion falls upon upper and lower cranial nuclei practically at once. The resulting picture is an aggregate of the simpler ones. From their vital nature, the presence of pneumogastric symptoms dominates the outlook.

Pseudobulbar Paralyses.—These are (1) organic and (2) asthenic. The organic variety is due to more or less symmetrical lesions involving the posterior group of cranial nerves at any point from their cortical centers to their peripheral trunks. We distinguish a *cerebral form* due to bilateral cortical or subcortical vascular lesions. The onset is abrupt, and usually developed in two stages. A hemiplegic or apoplectic case presents a second stroke, this time from a lesion in the sound hemisphere, and the bulbar palsy is at once established or completed. It is only very rarely limited to the parts definitely elected by true bulbar palsy, does not present the atrophy or degenerative reactions, and the reflexes are retained or exaggerated. A *radicular form* follows acute bulbar myelitis and hemorrhage into or softening of the bulb. These are rare affections of sudden onset. The lesion does not spare the motor tracts for the limbs, and other bulbar functions do not escape. Tumor may similarly produce a pseudobulbar palsy, but the distinctive symptoms of an intracranial growth—headaches, vomiting, vertigo, and papillitis—are added. A *basilar form* is occasioned by tumors of the base and basilar meningitis, especially of the syphilitic variety. A pseudobulbar paralysis of this form is among the greatest rarities, for obvious anatomical reasons. Finally we have a *neuritic form*. This is usually only a part of a more widely distributed or multiple neuritis.

Asthenic Bulbar Paralysis (*Myasthenia Gravis*).—This disorder, as described by Strümpell,[1] may mimic chronic polio-encephalitis inferior very closely. Previous to his communication a similar case was reported by Jolly,[2] under the title of "*Myasthenia Gravis Pseudoparalytica*," but is never so closely limited to the cranial nerves. Since that time cases have been reported by Murri,[3] Pineles,[4] Bruns,[5] Collins,[6] Hallervorden,[7] Kojewnikoff,[8] and many others.

The condition in several instances has terminated fatally, but no changes in the bulbar nuclei were discovered. Apparently the deficit or toxic effect had not reached a degree sufficiently intense to produce cell-changes that were observable under ordinary methods of examination. In a case reported by Widal and Marenesco,[9] disintegration of the chromophilic elements was demonstrated by the Nissl and Marchi methods. Goldflam [10] found widespread and decided changes in the muscles. This case and one reported by Long and Wicki presented preceding chronic pulmonary septic conditions. Laquer and Weigert have also noted widespread changes in the muscles apparently secondary to thymus disease. Laquer reports a case followed by progressive spinal muscular atrophy. Senator [11] suggests a relationship to various depraved blood states. Remak refers to a case of associated Graves' disease. Feinberg reports a case in which stercoremia was present and the asthenic symptoms retreated upon its relief. A persistent and enlarged thymus gland has been found so frequently, generally in association with widespread intramuscular infiltration of cells of a lymphoid character, that more than a casual relation seems to be implied between the myasthenia and the glandular state.

The paralysis, which comes on more or less insidiously, especially involves the tongue, lips, and pharynx, but the eyes and extremities are also affected to a certain degree. Ptosis is commonly encountered early. Usually, indeed, the myasthenia is general. Fibrillary twitching is wanting, and the reflexes are not disturbed.[12] Electrical responses are only modified by the fatigue induced by their repetition, presenting the myasthenic reaction, especially to the faradic current. It is found, as in bulbar paralysis, that rest seems to improve the paralytic features, but that the muscles involved show an extraordinary susceptibility to fatigue. Jolly's case showed ready muscular exhaustion under electrical stimulus, and this has been found in some other cases.[13] In some instances there is a tendency to improve and to relapse, as in the case of Collins, in which case the special senses of sight and hearing also showed rapid exhaustion.

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[1] "Deut. Zeit. f. Nervenheilk.," Bd. 8.

[2] "Berlin. klin. Wochens.," Jan. 7, 1895.

[3] "Policlinico," vol. ii, 1895.

[4] "Wien. Jahrbuch f. Psychiat.," vol. xiii.

[5] Schmidt's "Jahrbuch," 1896.

[6] "Internat. Med. Mag.," April, 1896.

[7] "Archiv f. Psychiatrie," vol. xxviii.

[8] "Deut. Zeit. f. Nervenheilk.," Nov., 1896.

[9] "Presse méd.," April 14, 1897.

[10] "Neurol. Centralbl.," Feb. 1, 1902.

[11] "Berlin. klin. Wochens.," 1899.

[12] Oppenheim. "Myesthenische Paralyse," Berlin, 1901.

[13] Buzzard, "British Med. Jour.," March 3, 1900.

The condition is marked essentially by asthenia, affecting particularly the motor apparatus. The prognosis is grave, as a fair proportion of the cases terminate fatally through asphyxia. In the treatment, rest and the free administration of strychnia are advised with correction of any toxic or septic factor that may be discovered.

PART III.

DISEASES OF THE BRAIN PROPER.

CHAPTER I.

THE CEREBRAL CORTEX—LOCALIZATION.

General Considerations.—The subject of localization of function in the cerebral cortex has attained great importance and its literature vast proportions. The practical considerations will be briefly and somewhat dogmatically set forth. Many points are still under debate, awaiting further experience and experiment, and some of these problems it is likely will never reach solution. It is well determined that there is a definite area of the cortex that is closely associated with motor functions. That this zone is purely motor is open to serious question. The general trend of belief is that it is also sensory, at least in part, and that motion does not even originate in it. As a working scheme we may consider that motion is represented in three levels: First, in the gray matter of the spinal cord; second, in the Rolandic area of the cortex; third, in the highest level of conscious thought, probably in the frontal region of the brain. The spinal level may be considered that of reflex, vegetative automatism, the Rolandic level that of motor memories, and the frontal area that of conscious, selective, and intelligent action. Thus, destruction of the highest level leaves automatic and memory action practically unimpaired, as in the experiments of Goltz, who removed the entire cerebrum of dogs without depriving them of muscular motion or bodily function. In the automatism of dementia the motor memories are likewise preserved. The mid-level, the Rolandic region, may be destroyed, leaving consciousness of volitional motions and the will to execute them, but the memory of their muscular production is gone, and they default, as, for instance, in motor aphasia. If the lowest or spinal level be destroyed, the mind and the memory organ have lost their tool and peripheral paralysis obtains. There is no difficulty in conceiving certain cortical areas to be memory organs, as in the case of the higher visual centers in the parietal lobe. We may, however, go further. All thought contains the two ideas of motion and sensation. They can not be separated, and without them consciousness is impossible. Indeed, they are in a certain sense identical. Motion is to the mind but the sensation of a change of position, and sensation is only the recognition of arrested motion. If, then, we consider the parietal convolutions as visual memory depots, we are equally at liberty to consider the Rolandic areas as motor memory depots. The flutist, by laborious conscious effort, establishes motor memories in his central gyri which can subsequently be called into operation by the will with a rapidity of which conscious thought is incapable. The new-born child can hardly direct its hand to its face, but very rapidly develops coördinate motor memories of this act in the motor cortex that thenceforward are easily recalled or subconsciously repeated. In the spinal levels single muscles or groups of muscles are represented. In the motor cortex coördinate and functionally associated movement memories are located, and in the highest level resides their volitional control and the power to recall and select them.

If these propositions are true in any degree, we would expect a neighboring relation of motion and sensation in the cortical representation, and this is no doubt the case. Horsley believes them to be superimposed in strata, (1) most superficially tactile sense, then (2) muscular sense, and finally (3) pure motor elements below. This is confirmed by clinical experience in cases of endarterial thickening, by which the blood-supply of the cortex is reduced. In such cases due to syphilis for instance, as the endarteritis comes on or recedes the sensory disturbance and paresthetic complaints are prominent. This is due to the arrangement of the cortical vessels. Arising from considerable arteries in the arachnopia, we have first a system of short capillary vessels that nourish the cortex, and especially its superficial or sensory layers, and, second, a system of longer and larger vessels that penetrate to the white matter without giving branches to the superficial cortex, but which do in part supply its deeper layers. It is at once evident that as the circulation fails the superficial, small, narrow, cortical vessels will first suffer. Hence we have sensory prodromata and sensory remnants in such diseases of the cortical arteries in proportion as, and because, the superficial or sensory strata of the cortex are mainly affected. There are numerous conflicting cases, ably marshaled in this country by Dana and Mills on opposite sides, in which cortical disease in the Rolandic area was or was not attended by sensory disturbance in the corresponding motor periphery. The writer has to declare that he has never carefully examined a case of gross disease of the motor cortex without finding some related sensory disturbance. It probably will be found temporary or permanent as it encroaches upon or affects the sensory layers of the cortex. It is more than probable that sensation is represented bilaterally in the cerebral cortex more completely than unilateral motion, and is consequently less modified by one-sided cerebral disease.

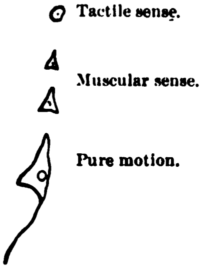

Fig. 66.—Horsley's view of the sensorimotor representation in the motor cortex.

The inharmonious views variously entertained relative to sensory depots in the cortex may be reconciled if we consider the sensorimotor zone as a midway station for sensation as well as motion and conceive of a higher cortical sensory level. There is a growing amount of material pointing to the parietal lobe as the seat of the so-called stereognostic sense, a complex faculty which is disturbed by lesions of the superior portion of this lobe, often with mind-blindness in association when left-sided.

The sensorimotor cortex is anatomically, or rather histologically, divided into from three to eight layers by various investigators. The important fact is brought out by all that the superficial layers are granular, and that the cells become progressively larger and more completely differentiated as we descend, until, in the lower strata, the cells correspond in appearance to the multipolar pyramidal elements of the motor horns in the spinal cord. The cortical cells are practically all present at birth, and the development and growth of the brain depends mainly upon the increase in their dendritic processes and the fibrous feltwork that supports them. It is now generally believed that the interrelation of these cells is due not to actual continuity of their processes, but merely to their interlacing and apposition. The conditions, apparently, which best favor the transmission of nervous influence and the functioning of nerve-cells would be close filamentous apposition. Withdrawal of contact might serve as an insulating and inhibiting measure, a theory strongly supported in some quarters, but as yet only a theory. The nuclei of these cells are now considered as only dominating their nutrition, and not otherwise essential to their activity, which is relatively the same at the dendritic periphery as in the cell-body.

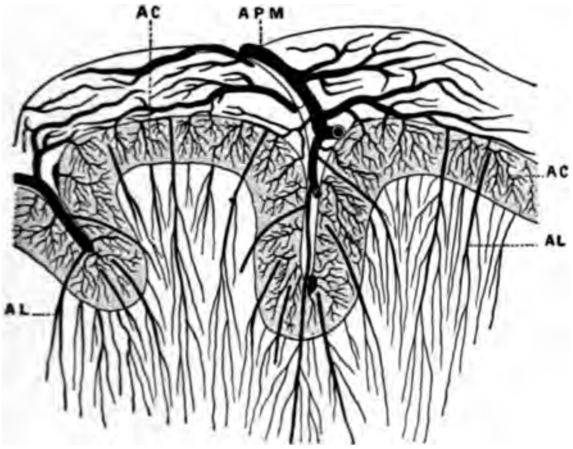

Fig. 67.—Scheme to represent the cortical arterial circulation. A P M, Artery of the pia mater; A C, short arteries to the gray matter only; A L, long arteries penetrating the white substance (after Brissaud).

Movements dependent upon paired muscles, such as those of the trunk, are rarely abolished by unilateral brain disease. Those of a specialized and one-sided character, however, may be completely inhibited by unilateral disease of their memory centers. Thus, the frontalis is rarely affected in cortical hemiplegia, while the unilaterally acting muscles of the lower part of the face are usually paretic in this condition.

It is an acceptable proposition that all skeletal muscular activity is bilaterally represented, and it is also true that all unpaired muscles and their coördinate activities are more particularly controlled by the opposite half-brain. The acquired faculty of speech, however, and the numerous motor and sensory memories associated with it, are almost always mainly represented in the left cortex in right-handed individuals. The superior weight and development of the left half-brain is probably largely attributable to its better nutritive supply through the arrangement of the vessels at the aortic arch and the larger caliber of the left carotid. This induces right-handedness, which in time, no doubt, retroactively increases the functional activity of the left cortex. At the same time the left hemisphere becomes potentially greater, more acquisitive, and therefore largely the seat of acquired motor and sensory education, which in turn increases its growth. It is probable that some overflow occurs in most brains, so that automatic emotional and expletive expressions either come to be located in the right brain or their frequent repetition sufficiently educates the right cortex to enable them to be recalled through its agency when the left centers are cut off. Similarly, if speech control be lost to the left half-brain, the right, especially in young persons, may be in turn educated to take its place in large measure. It is worthy of consideration whether the whole conscious and unconscious tendency of education, habit, custom, and practice to make all men right-handed is not a serious mistake. It seems plausible that should left-handedness, or rather ambidexterity, be assiduously cultivated in children, the two hemispheres of the brain might enjoy a greater equality and the individual secure not only amplified muscular control, but a certain lessened liability to aphasia and hemiplegic losses.

Motor Cortical Localization.—The human cerebral motor cortex has been mapped out with fair uniformity by numerous investigators. Following the lines laid down by Ferrier, Schaefer, and Horsley, originally based upon experiments on the monkey, the results of focal lesions in man and actual stimulation of the human cortex by electricity have given a fair degree of precision to the outlines of what I have ventured to call the motor memory area. Comparing figures 66 and 67, we may see that all skeletal-muscle groups are represented. Such outlines must be taken as suggestive rather than actual. There is no sharp boundary between the adjoining centers, and these fields overlap. The dippings of the sulci also serve to interfere with sharp limitations of the cortical areas and obstruct the experimental stimulation of individual movements. Every muscular movement, apparently, has a locus of principal or major representation in the cortex, but such a movement is so wrapped up with other coördinate movements, and so widely related functionally, that its representation in a minor degree may spread over great areas. The thumb, for instance, is principally represented in a given small cortical center, but the prehensile action of the thumb is related to the grasp of the fingers, the fixation of the wrist, the rigidity of the whole upper extremity, and even to action of the trunk and lower limbs in strongest efforts, during which the opposite members also come into play.

Beginning at the branching of the Sylvian fissure, we have, in the lowest and hindermost portion of the third frontal convolution, the movements of the *tongue*. Adjoining these caudad, on the lowest portion of the ascending gyri, we find the platysma represented with the *lower face* muscles, and then, above and on both sides of the fissure of Rolando, the *upper face* movements. In front of the ascending frontal gyre, occupying the rearmost portion of the second frontal convolution, is an area that is associated with conjoint movements of the *head* and *eyes* to the opposite side. According to Mott, stimulation of

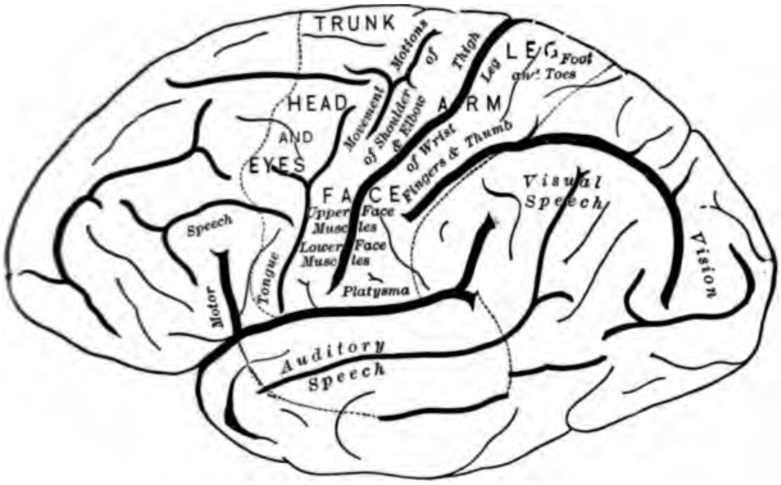

Fig. 68.—Functional areas of the cerebral cortex on the left side (Dercum).

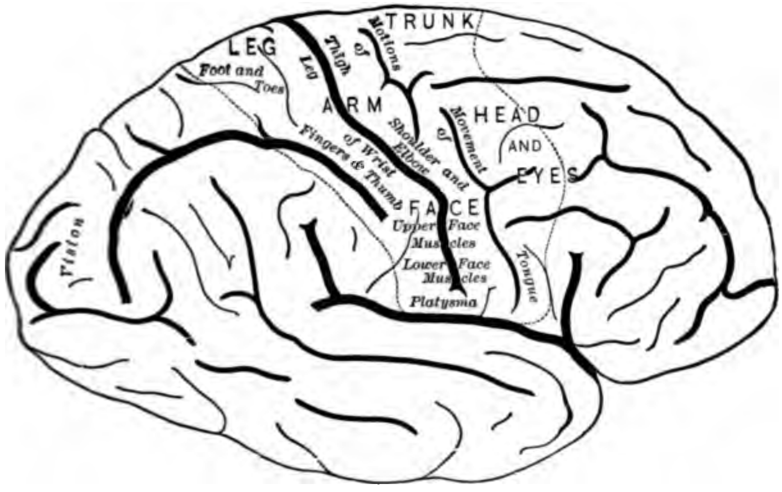

Fig. 69.—Functional areas of the cerebral cortex on the right side (Dercum).

the lower portion of this area in monkeys causes the head and eyes to turn to the opposite side and upward. Stimulation of the middle portion causes the head and eyes to turn in the horizontal plane to the opposite side, and stimulation of the upper third of this field directs the eyes and head to the opposite side and downward. It is probable that similar differentiation exists in the head-and-eyes territory in man. Roux, von Bechterew, and others assert that there is a somewhat similar center near the anterior margin of the occipital lobe, not distant from the visual speech memory depots, that also controls the pupillary and ciliary movements in accommodation (see p. 105).

Above the face area we find the *upper extremity* represented. This area occupies the middle portion of both ascending gyri and is bounded in front by the centers for the head and eyes. From below upward we note that the movements correspond in location to those of the opposite member as if it had been reached over the vertex and applied in foreshortened miniature to the cortex. Thus, in order from below and behind, upward and forward, we find successively represented the motions of the *digits*, thumb to the rear, *wrist*, *elbow*, and *shoulder*.

Lying above the upper extremity centers are those for the lower extremity. They occupy the caudad portion of the first frontal gyre to a slight extent, and the upper ends of both ascending convolutions at the vertical margin of the half-brain, over which they lap onto its median surface as low as the callosomarginal fissure. The *thigh* movements are anterior, followed in order, just as in the extremity itself, by those for the *leg*, *foot*, and *toes*, with the *great toe* last of all and to the rear.

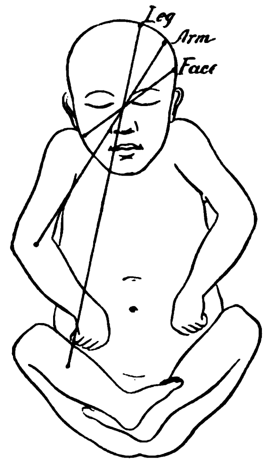

Fig. 70.—Relations of body to cortical areas.

Above the centers for the head and eyes, and in front of those for the lower extremity, are represented the *trunkal movements*. This area is relatively small, as these movements are simple and coarse in comparison, for instance, with those of the face and fingers. It occupies the posterior portion of the first frontal convolution, and laps over onto the median surface of the hemisphere. It is bounded caudad by the thigh area.

If we take the Rolandic area, from the superior margin of the half-brain to the Sylvian fissure, and divide it into fifths by horizontal parallel lines, the face occupies the lowest two-fifths, the upper extremity the next two-fifths, and the lower extremity the highest and remaining fifth.

Near the center for the platysma, which retracts the angles of the mouth, and in front of it, we find, in the lowermost portion of the ascending frontal gyre, the movements of *mastication*, and those for the *lips*, the *pharynx*, and the *larynx*. In front of this, in the third or lowest frontal convolution, or Broca's convolution, are located the memories of *motor speech*. These are confined to the left hemisphere in right-handed persons, and usually to the right half-brain in the left-handed.

Sensory Cortical Localization.—The impossibility of accurately determining sensory disturbances in animals is one of the major reasons for the confusion that exists regarding the cortical representation of this function. At present, however, no one claims that the motor cortical area is entirely devoid of participation in the receipt of afferent impulses. Mills reaffirms his belief that the limbic lobe and quadrate lobule are essentially sensory, and Dana admits that they may be so, but that the motor area is also sensory, or, rather, sensorimotor. The essential unity of motion and sensation has been already insisted upon in the general consideration of this subject, and the sensory mechanism in the superficial cortical layers has been pointed out. The probability of complete bilateral representation of sensation as a usual condition and the equality of the hemispheres in this relation have been adverted to. It would seem supposable that the sensorimotor zone may be an intermediate or memory depot for sensation, as it is for motion, and that there are higher psychical levels, which may be in the limbic lobe, as we have higher visual centers in the parietal lobe. At least we have the valuable clinical fact that in patients with brain injury the presence of stereoagnosis points to a lesion of the quadrate lobule above the area for visual speech. We should keep in mind that what is recognized as a sensation is the appreciation of arrested motion. We may thus conceive auditory sensation as the recognition, through the mechanism of the ear, of the arrest of sound-waves; visual sensations, as the recognition of the impact or contact with the retina of luminous waves, or light vibrations. The special senses of smell and taste are clearly dependent upon the contact of odorous quantities and sapid particles. It is always contact, always arrested motion, always touch. Regarding the location in the cortex of *general sensation* and its immediate modifications, the tactile, muscular, pain, and temperature senses, we are still in the mazes of speculation. The cortical relations of certain of the so-called special senses are more definite.

Vision.—The optic radiations arising from the outer geniculate body and in the neighborhood of the quadrigemina stream backward into the occipital lobe and reach the cortex at its apex. It may now be accepted that half-vision is represented at this location,—namely, that the corresponding half of each retina is subserved by the occipital cortex of the same side. The investigations of Henschen and others indicate that the macula is represented in each occipital cortex in more or less complete degree. It thus results that ablation or destruction of one occipital lobe produces double homonymous hemianopsia. In this condition the macula is regularly spared, as it is sufficiently supplied by the opposite lobe. It is likely that the half-retina may be further divided into irregular and varying upper and lower quadrants, with localized cortical representation. It is indicated by some clinical cases that the fibers in the optic radiation which serve the upper quadrants are above those for the lower, and that the macular fibers are placed between them. Altogether they make a bundle about a centimeter thick, passing horizontally backward at the level of the second temporosphenoidal gyre. They finally reach the apical occipital cortex, and seem to have their maximum field in the neighborhood of the calcarine fissure on the mesial surface of the lobe, over all of which half-vision is represented to some extent.

Higher visual coördinating and combining centers no doubt exist, and probably are in the angular gyre of the parietal lobe, where Ferrier first placed vision and where he still believes the macular representation is localized. This seems to be the seat of visual memories. Memories for printed speech are probably located here in particular and on the left side alone. Destruction of the left angular gyre, therefore, produces *word-blindness* and destruction of both angular gyri produces *mind-blindness*, all objects failing recognition.

Hearing is subserved by the first and second temporal convolutions, which are in relation with both ears. It, therefore, requires bilateral destruction of the gyri to produce complete *cerebral deafness*. Memories of spoken words are apparently stored in the posterior two-thirds of the first and second temporal gyri on the left side. When this area is destroyed, the right-handed patient becomes *word deaf*.

Smell and taste are presumably located in the cortex of the median surface of the temporal lobe, smell in the uncinate convolution, and taste below it in the fourth temporal. Broca, and after him Zuckerkandl, located smell throughout the limbic lobe, but Ferrier and later investigators are disposed to confine it to the uncinate gyre and the hippocampal region. The recorded cases bearing upon the locations of smell and taste are extremely few and not convincing. The region is not often affected by limited lesions. A few hemiplegics lose smell in the nostril opposite a lesion which involves the temporal lobe. Lesions of the tip of the temporal lobe have also been found in epileptics who had a gustatory aura or one of smell. Insane hallucinations of smell have been related to disease in this portion of the brain.

Cortex of Unknown Function.—Examination of the diagrams will at once indicate that the cortex of unknown function is much greater on the right than on the left side, owing to the fact that speech finds its representation almost solely in the left brain. In the prefrontal area, anterior to the so-called motor zone, it is customary to locate the higher psychical functions. While it is true that this region may be largely destroyed by injury or disease without producing localizing symptoms, there is a rapidly growing number of cases indicating that mental and moral obliquities are usually the sequence of such lesions. Ablation of the prefrontal region in dogs and monkeys induces a change of character, of disposition, of behavior, that is clearly recognizable. In men with prefrontal brain injury, mental sluggishness, want of attention, diminished memory, loss of energy and of self-control, are noted with more than coincidental frequency. The upper and posterior parietal regions, a portion of the temporal cortex, and the island of Reil are still unaccounted for. These are known as regions of *latent lesions*—of lesions which do not necessarily produce symptoms.

Craniocerebral Topography.—It is very necessary in many cases of brain disease to locate the underlying cortex, either for the purpose of operation or to determine the relation of scalp wounds, depressed fractures, and other traumata, to the cortex. No brain presents symmetrical hemispheres, consequently we can not expect a close resemblance between the brains of different individuals. There are also variations for sex, age, and body-weight. Very often there are supernumerary or unusual convolutions on one or both sides. A number of methods have been devised by Reid, Horsley, Hare, and others, to map out on the scalp the underlying cortical areas. They all have valuable features; but if you apply a number of them to the same head, so important a landmark as the Sylvian fissure will be variously defined. Absolute exactness, for the reasons already indicated, is not within the range of possibility. With the formulas named, the difficulty lies largely in selecting rather indefinite anatomical points, like the parietal boss or the temporal ridge, and making measurements in inches or other units from these points on ill-defined lines or angles. A simpler and probably more accurate plan is advocated by Drs. Anderson and Makins, of London, based upon a series of forty observations upon adult male and female heads and the heads of children. The system depends upon averages and proportions, making it of wider application than the others. It has served the writer well in many cases.

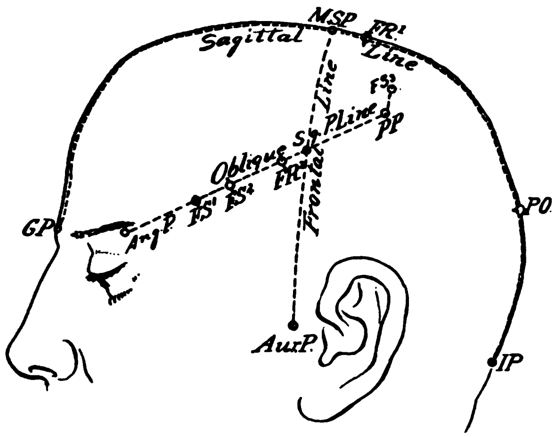

Fig. 71.—Craniocerebral guiding lines traced upon a cast of Professor Cunningham's (from a photograph). GP, Glabellar point, glabella opposite superior border of orbit; IP, inial point, at external occipital protuberance; MSP, mid-sagittal point, midway between GP and IP; Ang.P, angular point, external angular process opposite upper border of orbit; Sq.P, squamosal point, intersection of oblique and frontal lines at junction of middle and lower thirds of latter; PP, parietal point, termination of oblique line, equidistant with FS² from squamosal point; Aur.P, pre-auricular point, depression in front of tragus, at the level of upper border of external auditory meatus; FS¹, "commencement" of fissure of Sylvius, five-twelfths of distance from Ang.P to Sq.P; FS², bifurcation of fissure of Sylvius, seven-twelfths of distance from Ang.P to Sq.P; FS³, termination of fissure of Sylvius, $\frac{1}{3}$ of an inch above PP, in a direction parallel to frontal line; FR¹, upper extremity of fissure of Rolando carried to sagittal line in direction of fissure, $\frac{3}{8}$ of an inch behind mid-sagittal point; FR², lower extremity of fissure of Rolando carried to oblique line in direction of fissure, $\frac{3}{8}$ of an inch in front of squamosal point; PO, external parieto-occipital fissure carried to sagittal line in direction of fissure, seven-twelfths of distance from MSP to IP.

First a line, the *sagittal line*, is drawn in the median plane from the glabellar point to the external occipital protuberance. The *glabellar point* is determined by drawing a horizontal line at the level of the upper border of the orbital openings, and marking its intersection with the median plane. It corresponds very nearly to the union between the nasal and frontal bones. At exactly one-half the length of this line is marked a *vertical point*. Seven-twelfths of the distance from the vertical point to the external occipital protuberance, which can always be readily located, is a point corresponding to the parieto-occipital fissure, marking the limits of the parietal and occipital lobes. From the depressions immediately in front of the tragus of each ear, at the level of the upper margin of the external auditory meatus, lines called *frontal lines* are drawn to the vertical point already mentioned. At the junction of the middle and lower thirds of the frontal line lies the fissure of Sylvius, and this point, corresponding fairly well to the squamous suture of the temporal bone, is called the *squamosal point*. From the squamosal point a line, the *oblique line*, is drawn downward and forward to the external angular process of the frontal bone, at the upper orbital border level, as in fixing the glabellar point. Divide this line into twelfths. Five-twelfths of the distance from the angular point to the squamosal point begins the fissure of Sylvius; at seven-twelfths it bifurcates and extends by its horizontal line backward under the oblique line, and in its continuation to a distance posterior to the squamosal point equal to the distance of the point of bifurcation anterior to this intersection of oblique and frontal lines. At the termination of the oblique line the Sylvian fissure turns upward about ½ of an inch parallel to the frontal line, to terminate approximately under the parietal boss. To mark the fissure of Rolando, draw a line from the sagittal line ⅜ of an inch posterior to the vertical point downward and forward, at an angle of about sixty-seven degrees, to terminate ⅜ of an inch anterior to the squamosal point on the oblique line. The fissure of Rolando lies under this line, but terminates, as a rule, ¾ of an inch above the Sylvian, though very rarely opening into it.

Having thus fixed these two important and tolerably uniform fissures, the marking off of the principal convolutions is simple. Owing to the fact that there is no absolute relation between the cranium and the convolutions, it is necessary, when they are exposed, to verify them, if they are motor in character, by the application of the faradic current. This is done by means of an electrode bearing two probe-pointed terminals and may be accomplished through the unopened dura.

CHAPTER II.

SPEECH AND THE CORTEX—APHASIA.

General Conditions.—A word has four principal cortical qualities: it can be heard and seen, and it can be spoken and written. The first two are afferent qualities of perception. The second two are efferent qualities of expression. We, therefore, have four groups of word memories: (1) Those of sound, or *auditory word memories*; (2) those of sight, or *visual word memories*; (3) those of the motions necessary to express words in speech, *vocal motor word memories*, and (4) probably those of the motions required to express them by written symbols, *graphic motor word memories*. For these four memory groups we have four cortical memory depots, as indicated in Fig. 72. These memory word-centers are brought into mutual relations by systems of connecting fibers and into relation with their corresponding receptive and emissive peripheral organs by afferent and efferent tracts. We may indicate these connections by a diagram (Fig. 73). Thus, take the word apple; when it is spoken we hear it in the temporal lobe, and when written we see it in the angular gyre of the parietal lobe. We call up the memory of muscular movements in the third left frontal gyre when we would utter it vocally, and those probably in the neighborhood of the hand-center when we would express it by writing.

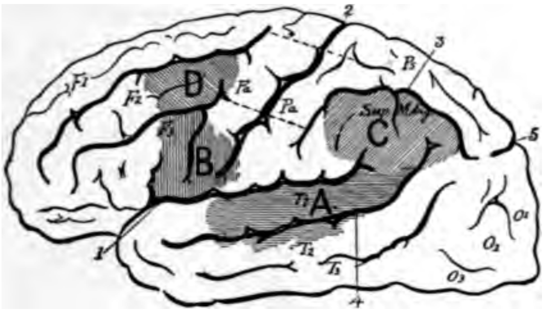

Fig. 72.—Speech areas in the cortex. A, Auditory word memories; B, memories for motor vocal speech; C, visual speech memories, including written characters and lip positions; D, graphic motor word memories (after Wyllie).

As one or another of these speech-centers is diseased we have corresponding varieties of cortical speech defect, or *aphasia*. They are: (1) Auditory aphasia, or word-deafness; (2) visual aphasia, or word-blindness; (3) motor aphasia, or aphemia; and (4) graphic motor aphasia, or agraphia. Disturbance of the connecting fibers also disturbs speech, giving rise to a number of *secondary* or *connecting aphasias*. Again, more than one center may be simultaneously affected, causing *combined aphasias*. The four primary word-centers are, to a varying degree, mutually dependent. Word-blindness is often attended by agraphia, as the mind is unable to recall the visual image of the word, which is to be copied, as it were, through the action of the graphic center. Again, if there is word-deafness, the patient can not write to dictation, though he may copy perfectly.

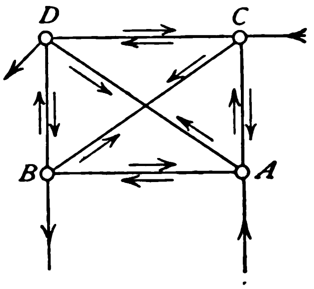

Fig. 73.—Diagram showing receptive and emissive speech-route, and the primary interrelations of the memory depots for speech. A, B, C, D correspond to the memory depots similarly indicated in the preceding figure (after Wyllie).

Individuals vary greatly in the local *storage* of their word memories. As first emphasized by Charcot, one may have his words principally stored in the auditory area (auditif), another in the motor area (moteur), and a third in the visual area. If we learn a new language by ear its memories are mainly auditory, and if by the book, they are visual. It is only when we have acquired oral proficiency that our motor memories are fully established and may finally, perhaps, predominate. Probably only those who constantly express themselves in writing for many years so fully establish the graphic motor memories that they become relatively independent of the other word-centers. It follows that children always have auditory word memories first developed, the motor or verbal expression following. The visual and graphic memories then in turn are acquired. From these considerations we can understand how it is that apparently identical lesions may produce results differing in degree in different cases.

The *stability of word memories* depends upon the intensity with which they are imprinted upon the convolutions, either by some special emphasis or by frequent use and repetition. Thus, words as well as incidents that come with the shock, for instance, of fright, are always readily recalled. Every one knows the value of repetition in fixing a point in the student's mind. Nouns, being least frequently used, are the most easily displaced, and proper nouns suffer the earliest of all. Verbs, adjectives, adverbs, and prepositions follow in a methodical order, governed by the rule of depth of imprint due to repetition in daily use. This necessarily varies with the individual. An oft-repeated oath, yes, and no are the most persistent of all.

It sometimes happens that the word or phrase on the patient's lips, at the moment of the stroke producing the aphasia, remains his only vocal expression. This he repeats at every attempt to speak, being, as it were, saturated or intoxicated with it. It has been suggested that its imprint under the circumstances of the stroke has made it indelible. In writing, the same thing is often indicated by the repetition of letters or words. The name is usually signed promptly, if the patient has been accustomed to the act, which has in time become automatic. Sometimes only the first few letters of a word are accomplished, and then repeated again and again, or words are repeated. The tendency to *echolalia* is also very noticeable, the patient repeating some word or expression he has himself uttered or just heard others employ.

In many cases of aphasia the patient miscalls objects or uses wrong words, especially names and personal pronouns. This is denominated *paraphasia*, and is most marked in lesions of the auditory word memory depot. In this case the patient does not make persistent attempts to correct himself, as he is unable to recognize his own mistakes. When the auditory centers are not primarily involved paraphasia is recognized by the patient, who constantly manifests his annoyance at being unable to utter the proper word.

The *mental capacity* of aphasics varies greatly, and must be carefully estimated in a given case. As a rule, it is somewhat modified, as is clearly indicated by the changed temperament and disposition practically always present, even in the slightest cases. The organic mischief producing the cortical lesion may induce complete dementia, and in any event the mind works somewhat clumsily. According to Bastian and others the sensory forms of aphasia, those due to auditory and visual disturbance, are more likely to present mental deterioration and to develop insanity than the motor varieties. In such cases hallucinations of sight and hearing, due to irritation of the cortex, are not infrequent.

Besides the loss of visual word memories, the patient is unlikely to recall figures, algebraic or other conventional signs, and musical notation. *Mind-blindness*, in which all objects are unrecognized, their visual memories being destroyed, is due, it will be recalled, to a bilateral lesion. In some cases of auditory aphasia the patient has shown a loss of musical appreciation, a condition termed *amusia*, and, as far as music is an acquired educated faculty, it is presumably associated with the left temporal lobe. Most motor aphasics whistle or hum tunes with more or less precision, and cases are of record where motor speech was lost, but the patient could sing the words of a song correctly. One case recently seen replied "God damn" to every question, but got out most of the words of "Annie Rooney" with distinctness when he attempted to sing.

Secondary means of expression, such as pantomime, facial expression, and gestures, are usually retained, but in rare cases the patient neither correctly produces nor understands them. This may go to the extent of nodding the head when negation is intended. The loss of gesture and mimicry is called *amimia ;* the misuse of these is *paramimia.* Emotional facial expression is generally retained, so that when the mind is not too much impaired the feelings of the patient are vividly portrayed in his countenance. There is some reason to locate these expressional centers in the basal ganglia, especially in the optic thalamus.

The motor cortex subserving speech is all within the domain of the middle cerebral artery, and aphasia is most frequently due to diseases of the branches or the trunk of this vessel. Trauma, meningitic disease, and new growths may also cause it. The most common simple variety of aphasia is the motor form, next the auditory, and then the visual. Distinct cases of graphic motor aphasia are extremely rare, but a convincing instance has been recorded. A combination of motor aphasia with the auditory and visual varieties is that ordinarily encountered. In this connection the various memory areas may be affected in different degree. No two cases in any one man's experience are exactly alike. Variations due to improvement or failure in the various speech qualities in any given case furnish constant study. It is important to know the initial conditions, when usually the picture is complete, in order to estimate the extent of the lesion. After a few weeks or months, through education of the opposite cortex and partial local recovery, the clinical picture may be entirely altered and quite deceptive.

Before taking up the various forms of aphasia the reader is referred to the methods of examination in such cases described in Part I, page 23. It may be well to call attention to the fact that the blind read type with their fingers, and that the deaf, by lip reading, cultivate special verbal memories, which must be specially localized in the cortex. We must also bear in mind that the word and the object are not the same, but that one is the symbol, sign, or name of the other. The wordpercept is, therefore, distinct from the object-percept, which is a part of the higher intelligence. It follows that the aphasic can mentally recall the object, as a chair, though he can not name it. The object-percept may also be called up by various routes besides those of hearing and seeing. By smell, or touch, or taste alone we can recognize familiar quantities that have corresponding qualities, and at once the word or name springs to the lips. The object-percept is, therefore, made up of and embraces all the attributes of the given object with which the individual is definitely acquainted.

Auditory Aphasia.—The lesion is in the left temporal lobe. The most striking condition is the *word-deafness.* The patient gets only a partial suggestion of what is spoken to him, or fails completely. When the intellect is fairly clear he is likely to make good guesses from the circumstances of the interview, the examiner's gestures, intonation, expression, or the motion of his lips. These sources of suggestion must be guarded in making tests. Usually, the visual speech-center, from its near location, suffers with the auditory, so that alexia is added, but if it escapes, the patient may read understandingly.

As most people are strongly *auditif,* the destruction of their auditory word memories notably impairs the emission of vocalized words. This manifests itself in paraphasia, and the speech may be reduced to a jargon or gibberish, which the patient expresses in a well-modulated way, and with an intelligent appearance. He does not recognize his own mistakes because of the word-deafness. He is likely to coin words and to repeat syllable sounds. Frequently he starts a word properly and then mutilates it. In trying to describe his feelings, such a patient said, with gestures to his head and abdomen, " I have a sulitar pretty well dear swell manœuver, and there are fullis things that hang solidar. There is a clean, fleshurable, pleasurable, fair, unsurizable, and any surizable way for a good deal insurations that is rotality all the time." He was annoyed that others could not understand him, and that he could not fully understand others. Very rarely would he attempt to correct a word. The writing of such a patient shows the same thing as his vocal utterances, both indicating the loss of the auditory speech memories upon which the majority of mankind are mainly dependent. He, therefore, uses wrong words, repeats words or letters, or his attempts lose all semblance to written speech. The ability to repeat words from dictation, or to echo sounds, may be destroyed, and is always greatly impaired. Copying written or printed characters is not affected. Amusia may be present.

Motor Aphasia, or Aphemia.—The lesion is in the foot of the third left frontal convolution, but usually extends to the foot of the ascending frontal, giving rise at the same time to motor difficulties in the lips and tongue. The characteristic condition in marked cases at first is the inability to produce articulate speech. Remnants like yes and no, or expletives, or an occasional habitual word may be retained, represented, as it is supposed, through the overflow into the right half-brain which occurs in the process of cortical education. As the right side is further educated, or if partial recovery in the left side ensues, the vocabulary increases. In the young the education of the right convolutions goes on with considerable rapidity if the mental impairment is slight. In recovering and in partial cases attempts at articulate vocal expression partially fail, from the loss of the memory of verbal motor combinations. In severe cases the condition is absolute. Motor aphasics sometimes misuse nouns (paraphasia), and they at once recognize their errors. They frequently paraphrase to get around their naming defect, as, for instance, "Give me some of that stuff for the bread," meaning butter. Wyllie also calls attention to the infantile characters of the speech in improving motor aphasia, such as lisping, lalling, and the cutting off of terminals and initial syllables. They can not repeat dictation any better than they can speak spontaneously. As a rule, agraphia is present and proportionate to the motor aphasia. For this a number of reasons are assigned. Usually there is right hemiplegia and the right hand is

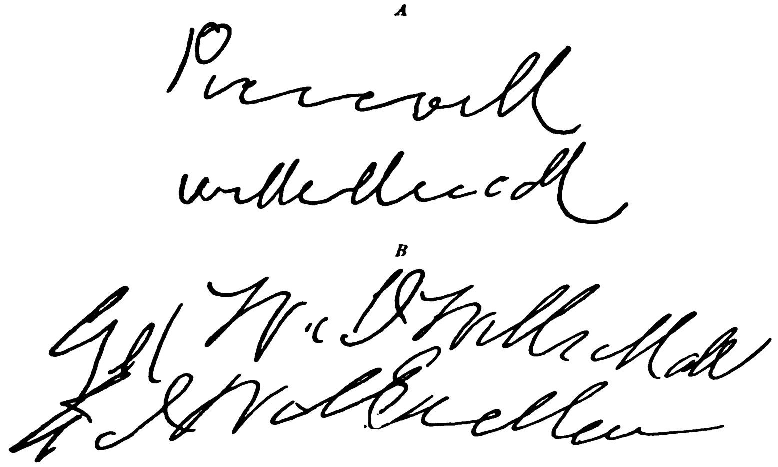

Fig. 74.—Handwriting in a case of motor aphasia. Patient's attempt to write his own name—Geo. W. Whitehall: *A*, spontaneously; *B*, after dictation.

powerless. When the hand is not disabled the agraphia persists none the less. It will be recalled that the graphic motor word memories are last acquired, the least deeply imprinted, and probably very easily disarranged. They are, no doubt, closely associated with the motor vocal memories, not only by nearness of location, but in function. As one learns to write, and even after much usage of the pen, he unconsciously inwardly pronounces first the letters singly and later the syllable sounds as he executes the written characters. A few rare cases of motor aphasia have retained fairly good use of written speech. These have presented subcortical lesions in some instances and cortical lesions in others. It may be that the individuals possessing deeply imprinted auditory, visual, and graphic-motor word memories would be less affected than the usual auditif-moteur class. In attempts at writing, motor aphasics frequently repeat a word or letter, or write only the few words of which they are vocally capable. Their names are usually signed with readiness if they can guide the hand. The motor aphasic who is able to use his right hand can copy readily and accurately, though he usually is unable to read. Even with the left hand they can copy as well as another. From dictation their efforts are no better than in spontaneous attempts. The motor aphasic understands all he hears and obeys commands. There is no word-deafness.

Reading is notably impaired and in the same degree as writing. The association of visual speech with motor speech is a close one. All in learning to read pronounce as they go along, and even in after life, when one is reading carefully, the tongue will be found slightly moving in the mouth in the same manner that the words would require were they pronounced. Yet many of these patients will intently scan the papers for hours or apparently read and reread the same books and insist that thay understand what they read. They almost invariably recognize their own names and in some cases seem to get the "drift" of what they read.

Motor aphasics showing a high degree of verbal difficulty are the ones in whom amimia and paramimia are usually observed. Occasionally they can not voluntarily protrude the tongue.

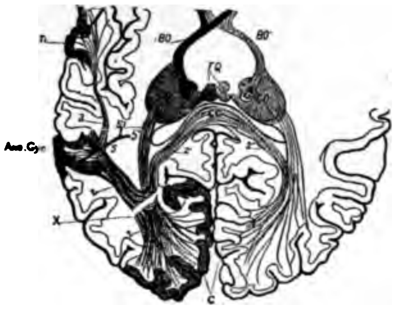

Fig. 75.—A lesion (X) divides the optic radiations within the occipital lobe, producing hemianopsia and word-blindness at once (Dejerine).

Visual Aphasia.—A lesion destroying the angular gyre on the left side produces the peculiar defect of visual aphasia, or word-blindness. If this lesion extends deeply enough to involve the optic radiations streaming from the basal ganglia to the occipital cortex, hemianopsia is added. A lesion in the optic radiation within the white matter of the occipital lobe may involve the connecting tracts between the half-vision centers in the apex and the higher visual centers in the angular gyre, producing both hemianopsia and word-blindness. It thus appears that word-blindness is due to disturbance of the angular gyre alone, and that associated hemianopsia is present only when the lesion implicates the optic radiation (see Fig. 75).

The visual aphasic can see perfectly anything put before him, but written symbols, figures, and other conventional signs have entirely lost their significance. That he sees them clearly is shown by the fact that he can copy or draw them with as much precision as ever. Spontaneous writing, however, is lacking. Not being able to call up the visual images of written speech, he is unable to exteriorize them through the motor writing apparatus, excepting in the case of some automatic combinations, such as his signature. For similar reasons he can not write from dictation. The hemianopsic cases, however, write spontaneously and from dictation with ordinary facility, as in these cases the visual word memories are not necessarily destroyed, but only cut off from the lower half-vision cortex in the occipital apex. They are unable, however, to read what they have written. Spoken language is both understood and expressed with complete readiness. When familiar objects are not recognized, mind-blindness is present and the lesion is probably bilateral. It appears that ordinary visual sensations, as sensation in general, are symmetrically represented. The educated faculty of visual word memories is represented in the left angular gyre alone.

Graphic-motor Aphasia.—Regarding this variety of aphasia there is much dispute and uncertainty. According to some, Wernicke in particular, the act of writing is but the act of tracing the visual word memories. He points out that this can be done with the left hand, the elbow, the foot, or even with a pencil held between the teeth. Exner maintains that there is a graphic-motor center in the foot of the second frontal convolution adjoining the centers for the hand, but this is not sufficiently verified. The case reported by Gordinier,[1] however, is very convincing. In this instance the circumscribed lesion, a small new growth, was located in the foot of the left second frontal convolution. The facility with which the right is used as compared with the left hand, or the other portions of the body, indicates that it has attained a proficiency quite its own, which must be resident in or near its principal cortical representation. Some persons write with small finger movements, others with a wide, full arm-sweep, the pen being simply grasped with the hand, which is largely guided from the shoulder. An ambidexter uses either hand. Their cortical writing apparatus must vary correspondingly. In persons deprived of both hands, who write with the foot, quite another cortical area must be educated for the purpose. It seems probable that in every case this education would fall upon the corresponding sensorimotor cortex, which develops specially intimate relations with the area of visual speech. Even this relation is not absolutely required, as the blind accurately reproduce the letter forms they have learned by the sense of touch.

It seems unnecessary to presume the existence of a higher graphic-motor center. A lesion which upsets the motor memories of writing would, therefore, probably paralyze the muscle groups used in the act, and it would be impossible to decide whether the difficulty arose from the obliteration of specialized motor memories or those proper to the member. In such cases the left hand writes as readily as ever if the visual word memories are not impaired.

It will be recalled that agraphia is common in motor and usual in auditory aphasia. In auditory aphasia we more commonly have paragraphia, unless the lesion also involves the visual speech-cortex. The usual proportionate relation of agraphia to motor aphasia early sug-

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[1] "Am. Jour. Med. Sci.," May, 1899; ibid., Sept., 1903.

TABLE OF SIMPLE APHASIAS.

VARIETY.	LESION LOCATED.	PRINCIPAL FEATURE.	UNDERSTANDS SPEECH.		EXPRESSES SPEECH.		REPEATS DICTATION.	COPIES WRITING.	WRITES FROM DICTATION.	INCIDENTAL CONDITIONS.
			Spoken.	Written.	Spoken.	Written.				
AUDITORY APHASIA.	Posterior thirds of the first and second left temporal gyri.	Word-deafness.	No.	Yes.	Paraphasia—jargon.	Paragraphia or agraphia.	No.	Yes.	No.	Amusia.
MOTOR APHASIA.	Foot of third left frontal gyre.	Dumbness.	Yes.	Partly.	Articulation impossible.	Exceptionally.	No.	Yes.	May do so.	Amimia or paramimia.
VISUAL APHASIA.	Angular gyre of left parietal lobe.	Word-blindness.	Yes.	No.	Yes.	No.	Yes.	Yes.	Partly, but can not then read it.	Homonymous hemianopsia, when the lesion is in the optic radiation; mind-blindness, if the lesion is bilateral.
CONDUCTION APHASIA (Wernicke).	Island of Reil usually, must cut off connecting tract between auditory and motor word memory centers.	Paraphasia. Paragraphia.	Yes.	Yes.	Paraphasia.	Paragraphia.	Paraphasia.	Yes.	Paragraphia.	Mental confusion.

gested to Broca and Trousseau that the motor speech-center must control this form of emissive expression as well as that by the oral apparatus. There is a general inclination to return to this view.

Conduction Aphasias.—The aphasias thus far considered are due to lesions of single memory depots. There are others due to lesions of the conducting tracts that bring these depots into mutual and coördinate relations. The most important is the one produced by breaking the path between the auditory and motor word-centers. This is usually due to a lesion of or in the neighborhood of the island of Reil. It was first described by Wernicke and is sometimes called Wernicke's conduction aphasia. These patients present no particular auditory or articulative difficulty, but, owing to the loss of correlation between auditory and motor memories, they lose selective ability when they try to express themselves, and a most marked paraphasia and paragraphia result. They are obedient to direction, but can not repeat dictation orally or in writing, though they copy with perfect precision. Neither can they read or pronounce aloud, though they seem to read understandingly. There is very little attempt to correct errors of spoken or written speech. The writer has seen such a case, due to traumatic hemorrhage, which was relieved by operation, the clot being found under the operculum on the surface of the insular convolutions, where it had been located from the symptoms. The recovered patient now states that there was considerable mental confusion during the aphasia, to which the verbal disturbance no doubt would conduce.

Wernicke, Lichtheim, Wyllie, and others describe four other varieties of conduction aphasia depending upon the location of the lesion in relation to the conducting tracts to and from the auditory and motor word memories. Some quoted cases also are given in their support. Anatomical findings as yet are scarcely adequate to firmly establish these varieties, which, however, serve to complete the pathological study of the subject. By their aid some rare instances can be deciphered.

Combined Aphasias.—It has been pointed out that all the word-memory depots are in the arterial territory of the middle cerebral. Consequently from this source, as well as by trauma, meningitis, or cerebritis, they may all be thrown out at once. Simultaneous injury to the auditory and visual word memories is comparatively frequent, and it has been seen that graphic-motor memories usually disappear with vocal motor memories—a combination due not only to association in function, but to proximity of centers, if a special one for writing is admitted. The loss of auditory and motor memories practically entails a loss of all speech attributes, as the visual and graphic-motor centers are so thoroughly dependent upon them. Again, the centers may be unequally affected, so that sensory disturbance preponderates over motor, or the contrary. The type cases indicated in the foregoing pages are indeed rare, but only by their full comprehension can we unravel the combined forms.

The mental disturbance is usually proportionate to the speech defect, and in total aphasia is very marked.

Reëducation of Aphasics.—One of the most important questions in a given case of aphasia regards recovery from the speech defect. Pure motor aphasia is perhaps the most hopeful variety in this respect, as verbal motor memories are the most easily built up. Reading usually follows much more slowly and writing is even more tardy. The forms of aphasia connected with the loss of sensory word memories are the most persistent. As often mentioned, a majority of persons are auditif, and all are necessarily so in childhood, though the child intently watches the lips of those teaching it to speak and probably acquires visual memories in association with the auditory impressions. In the sensory aphasias the mental disturbance is usually greatest. The loss of these earliest and usually most deeply graven memories, which become dominant in the speech mechanism, is the most difficult to overcome.

The first step is to determine by which route the intelligence may best be reached. Even when both auditory and visual memories are gone some patients can use the sense of touch to good advantage, and it will often be found an aid to put familiar objects into their hands when encouraging them to name them. If they are capable of giving attention, much may be expected. Simple sounds, such as a child first utters, like ba, pa, ma, may be indicated to them and repeated by the voice and in writing, by the position of the lips, mouth, and tongue of the instructor, or by taking the patient's hand and tracing the letters either in air or on a blackboard. If some object can be used,—say, a knife or pen,—it should be kept before the patient and placed in his hand when he attempts to name it. When simple sounds are mastered they can then be grouped into words, and the words associated with objects or actions, and so a vocabulary built up, which must be frequently and repeatedly and patiently rehearsed. By unremitting, intelligent effort, some emissive speech may be taught even the most hopeless cases. It adds greatly to their comfort and makes their care less burdensome.

CHAPTER III.

THE CEREBRAL WHITE MATTER, BASAL GANGLIA, AND CEREBELLUM.

Descending from the cortex in converging lines we have the *corona radiata*, the fibers of which bring the brain-mantle into relation with the lower brain parts and the spinal cord. Through the corpus callosum the homologous cortical elements on the two sides of the brain are brought into harmonious relation. The descending tracts reaching the basal ganglia are condensed into the *internal capsule*, in which the cortical motor fields are represented from head to foot, in an order from before backward, as indicated in figure 76.

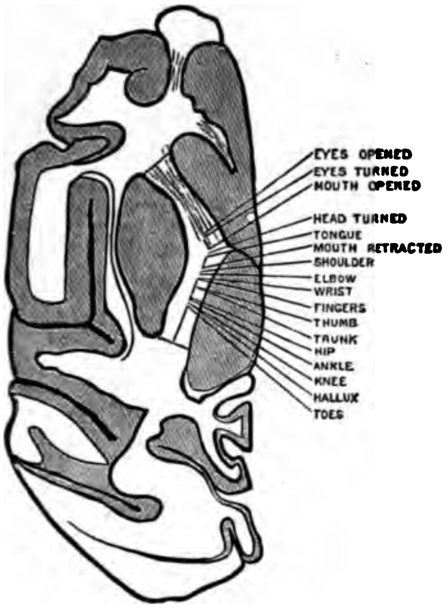

Fig. 76.—Arrangement of motor paths in the internal capsule (after Ferrier).

The internal capsule lies between the lenticular part of the striate body externally and the caudate nucleus and the optic thalamus on its median aspect, but is not dependent upon them. Its anterior portion, or limb, is supposed to contain psychic paths to the frontal lobes. Lesions of this part of the internal capsule produce no distinctive symptoms. The motor routes lower down are continued in the under portion of the crura and so on through the pons into the medulla and cord. The motor paths and their relations are indicated in figure 77, which shows that the face mechanism is inferiorly situated in the cortex, anteriorly in the capsule, and internally in the crus.

The *sensory paths,* situated in the posterior columns of the cord, pass upward into the posterior third of the hinder limb of the internal capsule. They reach the cortex of both hemispheres, according to Brissaud and others, directly on the same side and indirectly on the opposite side, by sending off a branching path through the corpus callosum, as shown in figure 78. Bilateral sensory representation is thus provided for. Doubtless motion is originally equally symmetrical in its cortical representation, the apparent functional difference arising from the specializing of unilateral motility in the opposite or most intimately related cortex, by practice, habit, and education.

From these diagrams we can understand that lesions in the cerebral white matter must be close to the cortex or in the internal capsule to produce permanent and definite sensory symptoms, as otherwise placed they do not intercept the pathways to both hemispheres. The symptoms in subcortical lesions correspond to the function of the related cortex, and in capsular lesions to the function of the particular tracts involved. The radiations to and from the cortex in the area of latent lesions may be, and often are, involved without giving rise to any symptoms.

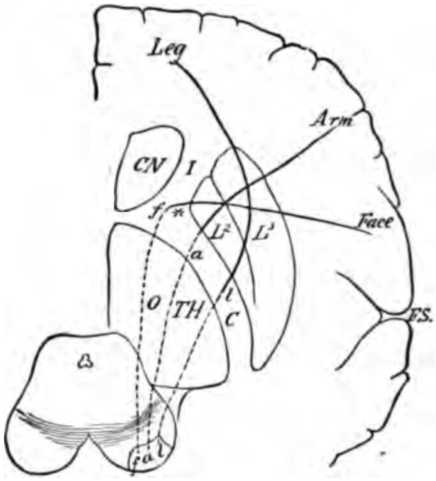

Fig. 77.—Diagram to show the relative position of the several motor tracts in their course from the cortex to the crus. The section through the convolutions is vertical; that through the internal capsule, I, C, horizontal; that through the crus is again vertical; CN, caudate nucleus; O, TH, optic thalamus; L^2 and L^3, the middle and outer parts of the lenticular nucleus; *f, a, l,* face, arm, and leg fibers. The words in italics indicate the corresponding cortical centers (after Gowers).

The *corpus callosum* may be diseased to almost any extent without presenting any peculiar symptoms. Bristowe, Sharkey, and Schuffer[1] contend that tumors of the corpus callosum are likely to produce hebetude, apathy, and prolonged placid coma. If its function is but to furnish correlating tracts between the two sides of the brain, its destruction would not materially interfere, perhaps, with the independent action of the half-brain. Putnam and Williams[1] note that in tumors of the corpus callosum mental changes are the only common symptoms, these consist in changes of character, impairment of memory and mentality, stupor, hallucinations, irritability and attacks of excitement. Epileptoid attacks, hemiplegia passing into diplegia most pronounced in the legs, and a peculiar ataxia, a sort of lack of balance, and a poverty of movement have been variously recorded.

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[1] "Rev. Speriment.," vol. xxviii, p. 2.

Lesions involving the optic radiations in the occipital lobes produce hemianopsia, and when, on the left side they cut the radiations from the occipital apex to the angular gyre, word-blindness ensues, as described on page 176. Lesions beneath the auditory word-centers likewise produce word-deafness.

The *corpora striata* can be completely destroyed on both sides without giving rise to motor or sensory disturbance if the internal capsule escapes. Their function is still a matter of speculation. Lesions affecting them, however, almost invariably implicate the capsular tracts. In this locality we encounter the hemorrhagic lesion that is preëminently causative of apoplectic hemiplegia. Whether or not posthemiplegic choreic movements and athetosis are due to disease of the basal ganglia can not be definitely stated, but lesions in this locality are frequently attended by such motor disorders.

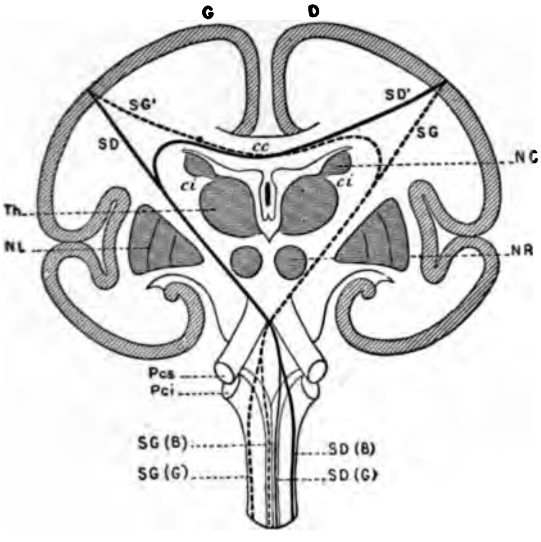

Fig. 78.—Schematic vertical transverse section of the hemispheres passing through the internal capsule and representing the sensory pathways. G, Left hemisphere; D, right hemisphere; *cc*, corpus callosum; *ci*, internal capsule; SG, sensory pathway from left side of cord; SD, sensory pathway from right side of cord. Both sides are brought into intimate relation through the corpus callosum, and the sensory representation is uniformly bilateral (after Brissaud).

The *optic thalami*, when diseased, give rise to no definite symptoms if the lesions are confined to their anterior portions and do not invade the capsule. When the hinder portion of the optic thalamus is involved we are likely to have either a crossed blindness or a double hemianopsia, probably from simultaneous injury to the optic tract. Bechterew claims, with much probability, that facial emotional expressions are controlled by the thalamus in some manner. Brissaud, in cases of postparalytic spasmodic crying and laughing, locates the lesion near the knee of the internal capsule. The loss of facial emotional expression at least points to the internal capsule and the optic thalamus rather than to the cortex.

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[1] "Jour. Nerv. and Ment. Dis.," Dec., 1901.

The *corpora quadrigemina* are seldom singled out by brain-lesions. When involved, adjoining structures almost invariably suffer, so that the resulting symptoms are difficult to analyze. The anterior pair are associated with vision, and apparently with some ocular movements. The pupillary reflex and movements of the eyeballs have been bilaterally impaired in some cases when they were diseased, and nystagmus, enfeebled vision, and blindness have been noted.[1] The posterior pair are thought to be related to hearing and equilibration. When they are diseased, the adjoining portion of the middle cerebellar lobe is usually involved, and probably gives rise to the symptoms mentioned. Raymond [2] contends that circumscribed lesions limited to the region of the corpora quadrigemina are prone to present (*a*) diminution of visual acuity, (*b*) palsies of associated ocular movements, (*c*) pupillary anomalies, (*d*) paresthesias on one or both sides of the body, (*e*) ataxic disorders of movement in the limbs, (*f*) athetoid and choreiform movements, (*g*) contralateral diminution of hearing, (*h*) rarely disturbance of mastication by implication of the motor root of the fifth cranial nerve.

The *crura cerebri* contain the motor tracts on their under portions and the sensory tracts above. Crural lesions, therefore, produce hemiplegia of the face and limbs on the opposite side, marked by hemianesthesia if the sensory paths are at the same time involved. The proximity of the third nerve, as it issues from the inner border of the crus, lays it liable to damage at the same time, and then we also have an ophthalmoplegia. This affects the eye on the same side as the lesion and opposite to the paralyzed limbs.—the Benedict-Weber syndrome. When the lesion is deep, it gives rise to nuclear disturbance, as described under Diseases of the Cranial Nerves.

The *pons Varolii*, when diseased, often presents characteristic symptom groups that make the localizing diagnosis comparatively easy. It will be recalled that besides giving passage to the pyramidal tracts, which course downward from the crura to the medulla oblongata, it is traversed by the root-fibers of the fifth, sixth, and seventh pairs of cranial nerves from their nuclear to their apparent origins. The course of the facial fibers and their decussation in the substance of the pons opposite the apparent origin of the fifth pair are described on page 119 and shown in figure 45. If a lesion falls upon the facial fibers before they decussate, and at the same time involve the pyramidal tract, which decussates lower down in the medulla, a paralysis of motion for the face and limbs on the side opposite the lesion ensues. If the lesion occurs below the facial crossing,—namely, in the lower third of the pons,—it will affect the face on the same side and the limbs on the opposite side, producing a crossed or alternating paralysis. Whether or not sensory symptoms are added depends upon the implication of the tegmental fibers, which lie above or behind the motor tracts. When this is the case the motor nucleus of the sixth nerve or its root-fibers is usually implicated at the same time, so that conjugate deviation of the eyes toward the side of the lesion and away from the paralyzed limbs is impossible. In destructive cerebral lesions higher up, it will be recalled, the ocular deviation is toward the lesion and away from the paralyzed limbs. Pontine disease may involve the motor speech-paths, which lie dorsad in the median portions of the pyramidal tracts, and give rise to articulative disturbance very like motor aphasia. Extension of the lesion dorsad and cephalad may involve the oculomotor centers.

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[1] Biancone, "Rev. Speriment.," Dec., 1899.

[2] "Lecons," Paris, 1901.

The motor and sensory portions of the fifth cranial nerve-root may be involved separately or together, and trismus may be induced by irritation of the motor nuclei. A lesion which cuts the sensory root-fibers of the fifth induces anesthesia in the face on the same side and crossed paralysis in the limbs through injury to the pyramidal tract. Rigidity, spasm, and choreoid movements in the limbs are sometimes encountered, and convulsions, in acute disease, are common. If the middle cerebellar peduncle is affected, vertigo, vomiting, and tinnitus are usually present, and deafness on the same side may ensue. Irritative lesions of this peduncle produce forced gyratory movements or forced one-sided positions in lying, which may be accompanied by corresponding positions of the head and eyes in the direction to which the turn is made. This may or may not correspond to the side of the lesion.

The *medulla oblongata,* owing to its small size and the vital importance of its nuclei, is most rarely invaded by acute disease without an immediately fatal termination. Disease of the olivary body may cut off the hypoglossal nerve and at the same time cause a crossed paralysis—the tongue on the same side and the limbs on the opposite. Diseases of this portion of the brain-stem are practically those of the lower cranial nerves and embrace the bulbar palsies already considered in Part II.

The Cerebellum.—According to Luciani, the cerebellum has for its main function the maintenance of sthenic tone in the muscular apparatus. If this be impaired, paresis, ataxia, incoördination, asthenia, tremors, and astasia result. It seems probable that the cerebellum is practically of a uniform functional quality, which is quite evenly represented throughout its entire bulk. One part may take the place of another. Risien Russell [1] and others have shown that the right lobe bears a certain relation to the right side of the body and the left lobe to the left side. Both sides are probably represented in the worm, or middle lobe. It is an experimental and clinical fact that cerebellar lesions of a sudden and extensive character at once produce very marked ataxic and paretic conditions, which may in time entirely pass away. Lesions similar or even greater in extent, but of slow development, may be entirely devoid of symptoms. It is evident that the cerebellum is capable of rearranging its functional relationships if gradually disturbed, and is of great recuperative powers after severe injury. Much confusion has arisen from confounding the symptoms of the secondary involvement of adjoining structures with those purely cerebellar.

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[1] "Brit. Med. Jour.," May 18, 1895.

We can now say that the right cerebellar hemisphere is in relation with the right side of the body and likewise with the left cerebrum. Mangazzini [1] has shown that injury to the thalamus induces atrophy of the opposite cerebellar half, and we thus have a crossed lesion involving the cerebellum on one side and the cerebrum on the other. With the thalamic lesion the corona radiata and sensorimotor cortex are usually involved.

A lesion in one lateral cerebellar hemisphere, if occurring with sufficient rapidity, as from hemorrhage or quickly developing abscess or tumor, produces *sthenic loss* on the same side of the body. This becomes manifest in one-sided muscular weakness or readiness of fatigue, in decreased coördination, in a tendency to stagger, and as the side of the lesion is the weaker side, the stagger is more marked in this direction,—that is, a patient with right-sided cerebellar disease is inclined to follow his right hand. Babinski,[2] under the name of *diadococynesia*, has particularized the difficulty presented by such patients in repeating a movement with rapidity and uniformity. This is commonly tested by asking for movements of pronation and supination of the forearm which quickly lose in uniformity and promptly subside from fatigue. The reflexes are also unilaterally reduced. At the same time the trunk may deviate to the sound side from the preponderating muscular tone on that side. Weakness of the ocular muscles on the same side as the lesion produces a tendency of the eyes to deviate in the opposite direction, and strong attempts to turn them toward the side of the lesion often develop nystagmic or jerky movements. It seems probable that lesions toward the head of the worm produce a tendency to fall forward, those toward the tail of the worm, a backward falling. These are the paretic manifestations. The cerebellar stagger and the ocular disturbance are often attended by *vertigo* of a pronounced subjective sort. Very commonly this is greatly intensified if the patient attempts to stand or even to sit up, and may prevent his doing so. In other cases, when the so-called cerebellar gait is well marked, there is no attending vertigo. Vertigo of a similar character may attend a tumor in the frontal region, which at the same time may cause an occipital headache and, according to Williamson,[3] in one-fifth of such cases induces bilateral weakness of the reflexes.

Irritative lesions produce another group of symptoms. They are marked by muscular stiffness in the extremities of the same side, by nystagmus, in which the jerk is toward the side of the lesion, and by such an arching of the body with the concavity to the diseased side as *tonic excess* on the affected side would produce. Emprosthotonos and opisthotonos would perhaps point to the middle lobe or to both lobes. Drummond[1] has also noted convulsions of a tetanoid character on the same side as the lesion, and Ferrier has recorded them in animals subjected to operation. The activity of the lesion dominates the symptoms. They grade off in proportion as the diseased process is slow and may easily reach a vanishing-point in chronic conditions that sometimes are astonishingly extensive.

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[1] "Neurol. Centralbl.," Aug. 1, 1895.

[2] "Rev. Neurologique," Nov. 15, 1902.

[3] "Glasgow Med. Jour.," Nov., 1899.

A third group of symptoms arises from *extension* of the cerebellar disease to neighboring structures, or from pressure upon them. A one-sided cerebellar tumor, for instance, by extension forward above the medullary decussation, presses upon the motor tract from the cerebrum to the cord and gives rise to spastic symptoms on the side opposite the lesion, with increased myotatic irritability and even a tendency to contractures. Pressure upon the floor of the fourth ventricle may affect the nuclei of the cranial nerves and give rise to paralysis of the fifth, sixth, seventh, eighth, ninth, tenth, and twelfth pairs of cranial nerves. The eighth or auditory nerve is particularly liable to be affected, and then aural symptoms are added. Tinnitus and vertigo of the Ménière variety may be superinduced, adding greatly to the complexity of the clinical picture. It is needful to investigate the aural condition very critically, as aural vertigo and cerebellar disease are often associated by the extension to the cerebellum of septic processes in the ear, and labyrinthine disease may closely imitate a cerebellar lesion. Irritation in the fourth ventricle may produce polyuria and glycosuria. Obstruction of the Galenic veins produces dropsy of the ventricles, their distention, and all the manifestation of intracranial pressure. Sudden death may follow disturbance of the pneumogastric nuclei. If the middle peduncle—the peduncle to the pons—be affected, *forced movements* result and *forced positions* are developed. These seem to be toward the opposite side if the lesion is irritative and toward the same side if destructive. Other clinical manifestations are those common to all intracranial diseases,—namely, headache, vomiting, optic neuritis, and vertigo. Of these an *occipital headache* is significant and is often associated with a rigidity of the neck and *retraction of the head.* Friedeberg[2] found this retraction marked in over half of the cases of cerebellar tumor in Aufrecht's clinic. Sensory disturbances are rare. Russell is inclined to think they may be present for a short time immediately after the onset of acute diseases, as hemorrhage, and transient anesthesia is noticed in operated animals. Krauss,[3] from a study of ninety-seven cases of cerebellar disease, enumerates the frequency of symptoms in this order: "Headache, vomiting, optic neuritis, vertigo, ataxia, asthenia, occipital pain and tenderness, inclination to turn toward the side of lesion, convulsions, and such secondary symptoms as nuclear paralysis, polyuria and glycosuria, tremors, and sudden death." Neither the mind nor the sexual desire is necessarily disturbed.

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[1] "Lancet," July 28, 1894. [2] "Berlin. klin. Wochens.," Aug. 19, 1905.

[3] "N. Y. Med. Jour.," June 1, 1895.

CHAPTER IV.

FURTHER LOCALIZING CONSIDERATIONS.

LESIONS of the brain may be broadly considered as *irritative* and *destructive*. From this point of view they respectively produce increased and decreased activity of function. We find the best exposition of these conditions in lesions of the sensorimotor cortex. Given a circumscribed definite lesion, such as a spiculum of bone or a small tumor that rather displaces than destroys the cortical elements in this region, and it is likely to so irritate them that increased activity is manifested in the peripheral area with which they are associated. A limited spasm or convulsion may ensue. If the irritation be too long maintained, necrotic or destructive cortical changes usually follow and are marked by diminished or completely lost peripheral function,—namely, paresis or paralysis. A lesion at first irritative may thus induce spasms in the hand, and after a time the hand becomes paretic if the progress of the disease reaches a destructive grade. Sudden destruction of the cortical mechanism, as by hemorrhage, causes immediate loss of power.

Were all lesions simply destructive or irritative, difficulty in deciphering them would be greatly reduced, but ordinarily they are combined in varying degree. Around a destructive process a zone of irritation brings new elements of disturbance into the symptom-field which, in turn, may be replaced by evidence of extending destruction. Again, in an area practically paralytic from cortical disease convulsions may occur, perhaps owing to irritation of remaining but inhibited cellular elements or from irritation of the subcortical tracts. In every case, therefore, it is highly important to know the clinical sequence of irritative and paralytic symptoms in order to determine the point of invasion, the progress of extension, and the limits of the lesion.

This brings us to the *invasion* or *extension symptoms*. These are transient in the widening convulsive manifestations of cortical epilepsy, and permanent in the slow encroachments of progressive disease. If we consider an irritant or discharging lesion to be located in a given part of the cortex, the disturbance to which it gives rise spreads in concentric and widening circles to the adjoining regions, which are successively upset, and the peripheral display is correspondingly and similarly broadened. The invading march of a Jacksonian fit can be foretold if we know its initial location or storm-center. Concentric rings on cortical diagrams enable us to grasp this point firmly. In figure 79, *A*, a fit starting in the arm-center would next call forth the face and head movements, then those of the trunk, and finally those of the lower extremity. Commencing in the lower extremity, the order would be reversed, as shown in figure 79, *C*. These sequences are in accordance with clinical facts. The order of convulsive invasion is not one of chance, but is rigidly dominated by the anatomical and functional relations of the cortex. The initial or *signal symptom* of a cortical fit, therefore, becomes highly significant as pointing to the storm-center, the point of greatest instability and usually the seat of organic disease.

Considerations of a similar character sometimes enable us, if we have all the clinical data available, to trace a neoplasm from its origin as it invades neighboring centers, and to relatively estimate its anatomical limits. The area of latent lesions must be kept in mind. It gives no special symptoms when traversed by the discharge of a fit or when invaded by a growth. The cortical fields of speech and the special senses are subject to the same rules as the sensorimotor zone. Their invasion is attended by aphasic or sensory disturbances. In the latter case hallucinations are likely to appear, and may constitute the signal symptom of Jacksonian fits. Thus, patients may always hear a certain sound, see a certain spectrum, smell or taste a certain article as the fit comes on. In at least one such case the application of concentric cortical lines showed that the subsequent motor disturbance was subject to the same invasion rule that obtains when the storm arises in the central convolutions.

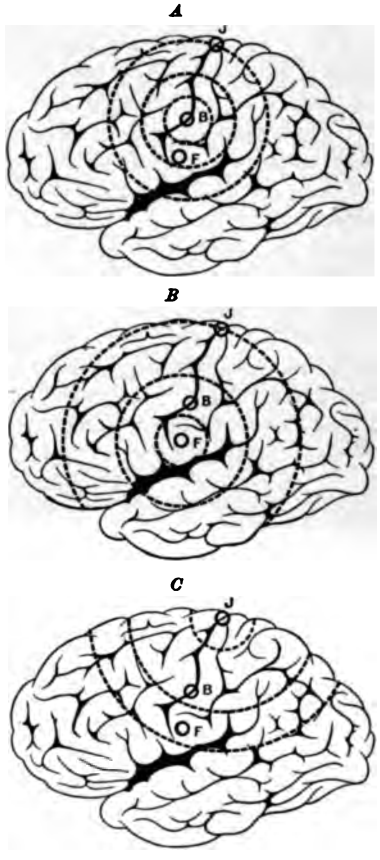

Fig. 79.—Schematic figures to show the encroachment of waves of discharge in the cortex, beginning respectively in the arm, face, and leg centers. F, Face; B, arm; J, leg (after Brissaud).

Peripheral *sensory disturbance* arising from cortical lesions is usually of a paresthetic sort. There may be some blunting of cutaneous, muscular, and joint sensations, but persistent anesthesia is extremely rare except in bilateral lesions. The double and complete representation of sensation has been sufficiently insisted upon (see p. 182). In Jacksonian fits the initial symptom is often a peculiar sensation or pain localized in or near the part that is first thrown into spasm. Patients are frequently at a loss to describe these sensations, and they vary from slight discomfort or slight formication to severe pain and intense burning. Their distribution is segmental or functional and does not conform to the peripheral nerve-supply. They are tolerably uniform in quality and distribution in any given case.

From another point of view cerebral symptoms are *diffused or local-*

ized. The great majority of endocranial lesions are marked by headache, vertigo, vomiting, mental disturbance, and often by optic neuritis. As these do not necessarily depend upon the part involved, they have little localizing value, but the presence of several of them is highly suggestive of brain disease.

Headache is a very usual symptom. It is pronounced in the cases which involve the meninges. Its importance in meningitis will be at once recalled. In syphilitic cerebral disease it has a marked tendency to come on toward night. Cerebral headaches are usually very intense and very refractory to sedatives. Lesions which do not impinge upon the cortex or meninges may give rise to no headaches whatever, unless pressure conditions arise, when the superficial portions of the encephalon are disturbed and pain ensues. Cerebellar disease is frequently attended by an occipital headache and a tendency to retraction of the head. The location of the headache is not always in close relation to the lesion. For instance, cerebellar disease has caused intense frontal headaches, and small tumors have given rise to a generalized head-pain. A circumscribed, deep-seated, persistent, and intense headache, however, has some localizing value.

Vertigo attends many brain disorders, and marks nearly all cerebral surprises arising either from without, as by concussion, or from within, as from embolism or hemorrhage. Implication of the aural apparatus gives rise to the peculiar vertigo described under Ménière's Disease (see p. 132). A lesion of the middle cerebellar peduncle causes forced lateral movements, usually associated with vertiginous sensations, and cerebellar disease is marked by a stagger, into the causation of which some vertigo may enter. In a general way we may say that persistent vertigo is likely to be allied to diseases of the basal parts. These, too, may give rise to the ocular and labyrinthine forms from injury to the motor nerves entering the orbit or vestibule.

Vomiting in encephalic disease is a common symptom. The peculiarities which mark cerebral vomiting are lack of gastric disorder and nausea, a clean tongue, and the readiness and ease with which the stomach rejects its contents. There is very little retching, and the food or drink is regurgitated, sometimes with considerable force, in a *projectile* manner. Meningeal invasions of an inflammatory or other character often present this symptom. It is very common in disease of the corpora quadrigemina, the pontine and the cerebellar regions, perhaps from more or less irritation of the pneumogastric nucleus.

All varieties of *mental disturbance* appear in organic brain disease. We encounter momentary unconsciousness in *petit mal;* hebetude, and profound coma in meningitis, apoplexy, concussion, and intracranial pressure; maniacal disturbance after epileptic attacks; changed temperament and character after frontal lesions, and more or less dementia in nearly all cerebral paralytics. The diffuse cerebritis of general paresis presents its own usually highly colored mental picture. The mental attributes of aphasics have been already described. Mental symptoms have the same general significance as motor signs. Irritation means mental excitement, and overmental excitement leads to hebetude, which also directly follows pressure conditions, shock, and deranged cerebral circulation.

Optic neuritis in brain disease has been adverted to under Diseases of the Optic Nerve (see p. 97). Its presence in suspected intracranial tumor almost serves to clinch the diagnosis. The location of a growth or the position of an abscess, or their size, seems to have very little to do with the intensity of the papillitis, but it is most common in lesions of the basal ganglia and cerebellum. When unilateral, the growth is commonly on the same side of the brain. A neuroretinitis is a common symptom and sequence of meningitis.

The *localizing symptoms* of brain disease have been set forth in the three preceding chapters. In addition we often derive much information from the implication of the cranial nerves that takes place in their intracranial course. The various symptoms thus produced are discussed under Diseases of the Cranial Nerves. Diseases of the base, the peduncular, pontine, and medullary portions of the encephalon ordinarily have cranial-nerve concomitants. This is also true of lesions of the cerebellar peduncles and of other parts in the region of the series of cranial-nerve nuclei.

Topical symptoms of some value are often encountered in intracranial disease, and should always be sought. *Traumata*, if recent, are usually marked by bruises, wounds, or fractures that at once center attention on the underlying parts and on the opposite side of the brain where the force of the blow is expended. In later cases scars, cranial depressions, or evidence of bone disease are equally significant. The presence of *deformities* due to new growths and the conditions of the auditory canals, nasal passages, and pharyngeal vault are to be carefully noted. When the disease affects the meninges there is often topical *pain* and *tenderness* that can be elicited by making pressure over the scalp or by going over the surface with a percussion hammer. Its outline and permanence are suggestive of the extent of disease beneath. A neuralgia of the fifth or occipital nerve has its own tender points and anatomical outlines to distinguish it. By *percussion*, Macewen, Starr, and others have been able to distinguish a different note over the seat of intracranial growths and diseases that were located close to the cranial wall. This, doubtless, requires a very acute ear, but should be sought in every suspicious case. Intracranial aneurysms may in some cases present a *bruit* that can be heard through the stethoscope. We would naturally expect the patient to be aware of it, as the conditions favorable for bone-conduction would be present. Two of the writer's cases of extensive intracranial aneurysm, in which a bruit was probably present, gave expression to no such subjective complaint, and auscultation was not attempted before operation. In a third, persistent throbbing had annoyed the patient for months and then disappeared, but the stethoscope revealed a decided bruit, of which she was not conscious.

In some cases of intracranial disease a *localized elevation of temperature* has assisted the diagnosis. Our present commonly available means of surface thermometry are deficient in accuracy. If the bulbs of clinical thermometers be passed through pieces of rubber protective and then applied to the scalp by light bandages, tolerably reliable readings may be obtained. It must be borne in mind that the left side of the head is usually, in the right-handed, about one degree warmer than the right side, and that mental activity causes the temperature to go up nearly or quite another degree. Sometimes in suspected abscess a thermometer in each auditory canal may on one side show local heat even in the absence of any apparent local inflammation. Palpation may discover a brain tumor, as the cranial wall is often eroded by a cerebral growth, and that, too, when situated at a considerable depth. The *x-ray* has been used in many cases of brain disease. In a few instances it has aided in the diagnosis of tumor, but in cases of exostosis, foreign bodies, and hemorrhage, skiagrams are of the greatest value.

Finally, it is to be borne in mind that a brain-lesion located in a latent zone may give rise to *symptoms at a distance*. In some instances this results from interference with the blood-supply or the return circulation. In other cases the mechanism can not be explained. In many cases where every symptom pointed to the cortex, the disease has been found deeply seated. The localizing diagnosis always contains uncertainties and should be expressed with a fair degree of reservation. Operations depending on it are, therefore, exploratory in every instance when external guiding signs are lacking.

CHAPTER V.

ARTERIAL BRAIN DISEASES.

IN the preceding chapters of this part the localizing features of brain disease have been considered. They furnish the basis of the *localizing diagnosis.* An equally or more important question is that of the *pathological diagnosis.* What is the lesion? Comparatively few pathological processes are found in diseases of the brain, but their effects and results are numerous and serious. These diseases fall into groups related to: (1) The arterial supply; (2) the venous return; (3) inflammatory disturbances of the brain-substance, and (4) new formations. Surgical conditions, such as penetrating wounds, are left to works properly covering such accidents. We first turn our attention to the arterial supply of the brain.

Anatomical Considerations.—The arterial supply of the encephalon furnishes the anatomical basis of some brain-lesions, and presents practical points of great importance. The left carotid, leaving the arch of the aorta on a tangent that conforms to the natural blood-current, favors the passage of emboli of cardiac origin to the left brain. It will be recalled that all the blood to the brain proper reaches the encephalon by the internal carotids and the vertebrals. These four inlets are brought into intimate relation through the circle of Willis. From side to side compensation in the circle is practically complete in case the lateral arteries are occluded. From the carotid or anterior portion of the circle to the vertebral, basilar, or posterior portion, full compensation can not be provided owing to the smallness of the connecting arteries. From the circle of Willis two distinct systems of arteries arise,—the basal and the cortical. From the circle itself, and from about the first inch of the six great arteries, the anterior, middle, and posterior cerebrals, short, direct vessels plunge into the brain to nourish the basal parts, ganglia, and capsules. These anastomose but slightly with one another, and are of the nature of terminal arteries. Their occlusion or destruction irreparably cuts off the circula-

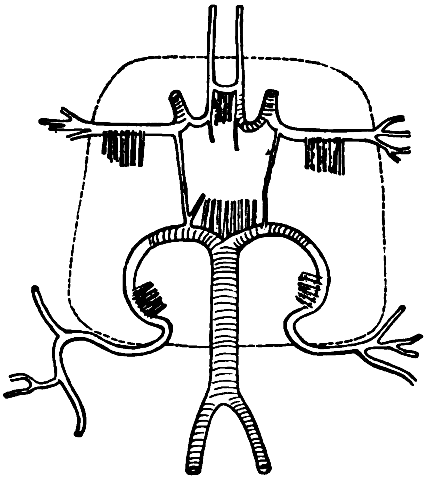

Fig. 80.—Charcot's diagram of the circulation at the base of the brain.

tion from a given portion of brain-tissue. Those distributed to the lenticulostriate nucleus, the internal capsule, and a portion of the thalamus are derived from the stem of the middle cerebral. One in particular, supplying the third layer of the lenticular ganglion, the caudate nucleus, and the upper portion of the capsule, is so commonly the seat of rupture that it was denominated by Charcot the artery of cerebral hemorrhage.

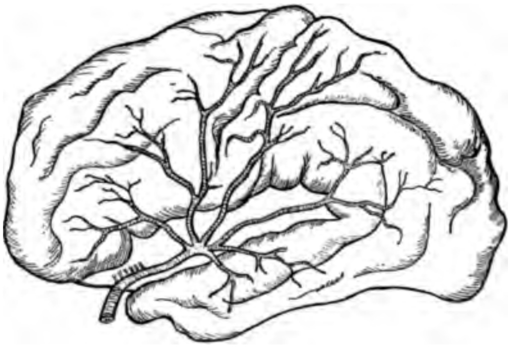

Fig. 81.—Distribution of the middle cerebral artery (Duret).

The main cerebral arteries coursing up over the hemispheres in the arachnopia have each a definite cortical territory, and these only slightly overlap. They give off two sets of branches,—namely, delicate arterioles, nourishing the cortex, which they enter at right angles, and larger straight, long branches, which pierce the cortex and supply the white matter of the cerebrum (see Fig. 67, p. 163). These pass inward and almost come into anastomotic contact with the upward-reaching terminations of the capsular and ganglionic branches arising close to the circle. Between these arterial territories there remains an ill-nourished zone that is prone to senile softening (Fig. 83).

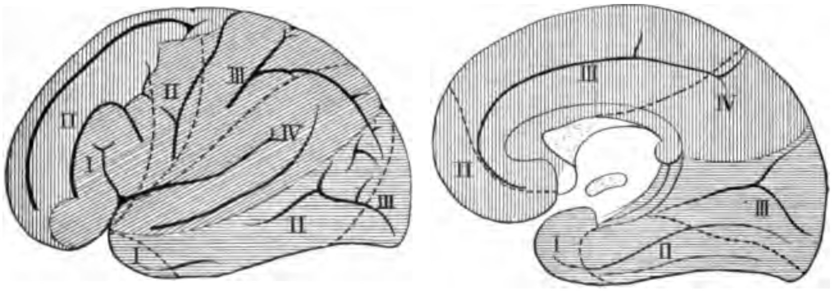

Fig. 82.—Showing the distribution of the anterior, middle, and posterior cerebral arteries on the surface of the brain. The numerals I, II, III, IV indicate the areas supplied by the different branches; the dotted lines indicate the main trunks (modified from Merkel and Debierre).

The cerebellum receives its blood-supply from the vertebrals and basilar; the medulla and pons are largely supplied by the basilar. It will be recalled, from the description of the pial vessels (p. 72), that their cortical and deeper branches carry with them a periarterial lymph-sheath continuous with the pial space.

CEREBRAL ANEMIA AND HYPEREMIA.

Cerebral Anemia.—Until recently an undue importance attached to the frequently alleged conditions of general brain anemia and hyperemia. They were spoken of as morbid entities, and diagnosis stopped at that. We must look upon them as purely symptomatic and secondary. In many respects they are marked by similar or even identical phenomena, and can only be distinguished by their associated states or incidental symptoms. The circulation of the brain, it will be recalled, is under the direct control of vasomotor centers in the medulla and cervical cord. It is exceedingly difficult, if not impossible, to say where physiologically increased blood-supply becomes a morbid congestion and entitled to the term hyperemia. Cerebral anemia is equally indefinite. In giving the causes of these opposite conditions, therefore, it has been found impossible to sharply separate the natural from the morbid states. Let us first consider the anemic brain.

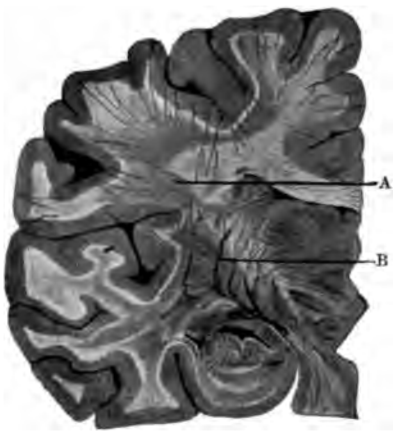

Fig. 83.—Scheme showing the anastomotic relations of the basilar and cortical arterial supplies. A, Common location of senile softening; B, common location of cerebral hemorrhage (after Brissaud).

Etiology.—Cerebral anemia is most common in infancy and old age, the periods of least brain activity. In old age it is the legitimate consequence of senile arterial degeneration. In adults it follows intense emotions, various peripheral, acute, painful disturbances, and shock of all sorts. Tobacco, ergot, belladonna, bromids, antimony, lead, chloroform, and many other drugs and poisons produce cerebral anemia. Fatty heart, aortic insufficiency, bradycardia, excessive hemorrhage, rapid evacuation of peritoneal and pleural effusions are attended by cerebral anemia. It takes a prominent place in all the general anemias, and in the pernicious varieties is especially marked and often attended by nutritional changes in the gray matter of the brain and spinal cord. Cachectic and exhausting diseases, such as cancer, typhoid, phthisis, etc., produce a similar condition. Ligature or obliteration of a carotid, or partial occlusion of both carotids or vertebrals by atheroma, serve to mechanically produce anemia of the brain.

Symptoms.—In *acute forms* of brain anemia, such as are typified by an ordinary fainting spell or a great loss of blood, the symptoms are "darkness before the eyes," giddiness or vertigo, noises in the ears, feebleness, trembling of the limbs, nausea or vomiting, sometimes slight delirium, and then more or less loss of consciousness. Partial or generalized convulsive movements are common, and epileptiform convulsions are sometimes seen. Finally the condition remits or passes deeper into coma, and may terminate in death. The face is usually blanched, the pulse small and fluttering, the pupils dilated, the skin covered with a cold perspiration. Similar conditions are seen in shock, and to a moderate degree in some cases of migraine.

In the *chronic form*, such as attends general anemia and the cachexiæ, patients complain of heaviness of the head, of headaches limited to a portion of the head, or a feeling of constriction about the head. They sleep lightly or brokenly and their sleep is disturbed by dreams. There is mental and physical inertia. They are irritable, peevish, impressionable, and somnolent. The pupils are dilated. Vertigo, sometimes ringing in the ears, and muscæ volitantes are common, and often induced by rising from the recumbent position or by a quick turn of the head. In extreme cases there may be visual hallucinations and temporary blindness from retinal anemia. Slight delirium or maniacal states and even epileptiform convulsions are seen in severe instances. Optic papillitis or choked disc is occasionally encountered.

The **diagnosis** is not difficult except in the toxic cases. It must be remembered that the circulation in the face, or even in the retina, is not a reliable index of the brain state, but is sometimes a helpful guide. Anemia of the brain is relieved by placing the head low and intensified by the vertical attitude.

The **treatment** is that of the underlying causal state or toxic condition.

Cerebral hyperemia, like anemia of the brain, is always a secondary state, and, while arising from opposite conditions, has many similar subjective symptoms. It may be recalled that the passing from sleep to the waking state is normally marked by increased cerebral circulation, as is also the process of active mentation, which is attended by a slight increase in the surface temperature of the head, particularly on the left side in right-handed persons. The brain volume is augmented and intracranial cerebrospinal fluid is displaced in cerebral congestion.

Etiology.—Muscular effort, coughing, vomiting, increased heart action, impeded respiration, a dependent position of the head,—all tend to increase the amount of blood within the skull and produce congestion or hyperemia of the brain. Usually the face shows a similar condition. Some families display a tendency to plethora and cerebral congestion. Menstrual periods are normally marked by an increased circulation in the brain. At the climacteric this occurs in flushes or waves and becomes very annoying. Gouty subjects are particularly liable to vascular disturbance, which frequently involves the cerebral circulation. Contracted kidney and arteriosclerosis are also commonly marked by such vascular storms. Insolation produces intense cerebral congestion, and exposure to cold has a similar effect by driving the blood from surface to center. In the same way during a chill the central organs are hyperemic. The nitrites, opium, alcohol, and the infections of typhoid, pneumonia, tetanus, and many fevers produce cerebral congestion. This may alone be the cause of the convulsions which so frequently mark the onset of eruptive fevers in children. The suppression of menstrual discharges or of chronic hemorrhoidal bleeding and pressure on the aorta by a tumor or fecal accumulation raises the cerebral blood-pressure. Cardiac conditions may cause cerebral congestion by impeding the venous return or increasing the arterial tension. Inflammations about the head and neck and in the throat are commonly attended by cerebral congestion. Brain-tumors and continued epileptic attacks usually cause increased cerebral vascularity.

Symptoms.—*Acute cerebral congestion* of a marked degree usually causes intense throbbing headache, haziness or blurring of vision, and sometimes photophobia. It sometimes leads to vertigo, ringing or throbbing in the ears, a tendency to somnolence, and if sleep ensues, frightful dreams are likely to disturb it. There is mental depression and incapacity for thought.

Acute cerebral congestion in a *grave form* occasionally comes on like an apoplectic stroke and furnishes an *apoplectic equivalent.* The patient falls inert, unconscious, with stertorous respiration, relaxation of the sphincters, and may die. He usually recovers, however, in a day or two. Slight paralytic symptoms last several days longer and eventually completely disappear. All gradations of cerebral congestive attacks are encountered, and, as above indicated, it is often difficult to distinguish at what point they become pathological.

In gouty cases and in various chronic intoxications there is a tendency in some to delirium, in others to convulsive manifestations, during the attack of cerebral hyperemia. These motor and mental excesses may reach epileptoid and maniacal stages.

Chronic hyperemia of the brain is usually a part of organic disease of that organ and does not here call for special mention, as its symptomatic value is commonly apparent. When it gives rise to symptom groups it is by exacerbation in the form of acute attacks.

The **diagnosis** of cerebral congestion is commonly easy. The feeling of fullness in the head, the injected eyes, the flushed face, full arteries, quickened pulse, contracted pupils, and the increase of all symptoms when the head is lowered are sufficiently striking. It is often very difficult, however, to distinguish the epileptoid and apoplectiform attacks from those of pure epilepsy and gross brain-lesions respectively. In the case of apoplectiform attacks in plethoric individuals before the age of fifty, especially if they are alcoholic or gouty, the presumption is in favor of cerebral congestion and reservation of opinion is in order. In epileptiform attacks the history of the case will almost invariably illuminate the situation.

The symptom, hyperemia, having been deciphered, it remains to trace it to its proper source, and to this treatment is directed. Repeated cerebral congestive attacks in elderly persons foreshadow cerebral hemorrhage and softening. In paretic dementia they are likely to be followed by apoplectiform attacks and a rapid downward course. In other instances their significance is strictly related to their cause.

CEREBRAL ARTERITIS.

Immense importance attaches to disease of the cerebral arteries, not so much on their own account as because of the dire consequences of hemorrhage and softening which they may entail.

Acute arteritis in the brain may follow infectious diseases, such as typhoid, variola, diphtheria, scarlet fever, measles, and puerperal infection. All the arterial tunics are involved, but distinct symptoms do not arise unless thrombosis ensues. This is particularly liable to occur, as the intima is often much thickened. The arterial inflammation in many instances arises first in the vasa vasorum, and is thence propagated to the arterial trunks. The softening that ensues may, according to Turner, be followed by cerebral hemorrhage.

Peri-arteritis is a descriptive term applied to a proliferating affection involving the external arterial coat. It is attended by diffuse or circumscribed thickenings and connective-tissue increase. The perivascular sheaths of the cerebral vessels become choked. This, taken with the weakened arterial wall, favors the formation of saccular dilatations. In many instances the cerebral vessels become beaded with minute aneurysms, which are prone to develop at the branching points. In themselves these *miliary aneurysms* favor rupture, and the fatty degeneration of the arterial and capillary walls increases this danger. As a fact, they are the almost invariable source of cerebral hemorrhage, and can usually be found by carefully washing out the clot.

Peri-arteritis is frequently caused by, or at least associated with, Bright's disease. It may be induced by tubercular infection, which usually invades the cranium by the arterial route. It is a concomitant of the involution of advanced years and of arteriosclerosis. Syphilis may produce it, as may gout, rheumatism, and alcohol. Symptoms are very vague and indefinite, or absent, until hemorrhage or infarction gives rise to plegic disturbances. The condition should be suspected in chronic Bright's disease and in cases showing arterial degeneration elsewhere.

Chronic arteritis, *atheroma, endarteritis deformans,* is frequently found in the large cerebral vessels, particularly those at the base, and especially the basilar. The atheromatous plaque originates in the occlusion or inflammation of the nutrient artery, or vasa vasorum. This produces an infarct largely confined to the middle tunic of the vessel, and the fatty degeneration that ensues is eventually replaced by calcareous deposits. The vessel may be completely encircled by such a patch, or numerous atheromatous islands may be found. By their coalescence the entire artery becomes rigid and brittle. At first the intima covers the plaques smoothly, but it is prone to break down, leaving the calcareous matter exposed in the blood-stream. This frequently leads to local deposits of fibrin from the blood, which may cause thrombotic closure of the vessel or wash away in embolic masses or particles, to produce disturbance farther along the course of the arterial current. In the same way calcareous particles may be cast into the stream, and, lodging in the narrowing channel, cause secondary mischief. Another effect of atheroma is to narrow the lumen of the affected vessel through the thickening of its walls and the swelling of the internal coat. Again, by weakening the vessel-wall, atheroma may produce dilatation and lead to an aneurysm. The small arteries arising at the seat of atheromatous invasion may be occluded, though the parent vessel remains pervious. Collateral territories are thus cut off, while the ultimate distribution remains active. The rigidity and brittleness of the artery favor rupture and hemorrhage.

Atheroma may be considered as a purely senile condition in many cases, a part of the involutionary changes of the organism in advanced years. There is no doubt that gout, rheumatism, great muscular straining, overindulgence in alcohol, lead poisoning, and syphilis are additional causes. Lancereaux says chronic malarial infection may cause atheroma. Causes are frequently combined in a given case, as gout and senility. Atheroma, though usually found after middle life, has been noted in the aorta and large systemic vessels in children and even in infants.

The symptoms of atheroma of the cerebral vessels are usually vague and uncertain until thrombosis, aneurysm, or hemorrhage has been added. It may be reasonably suspected when the condition in the heart, aorta, and palpable systemic arteries indicates its generalized distribution. Protracted nosebleed after middle life is generally due to arterial degeneration of carotid branches in the nasal spaces and a rather common forerunner of cerebral arterial accident.[1] Lesion of the optic chiasm by bilateral atheromatous thickenings of the carotids pressing upon it has been noted. Double temporal hemianopsia may thus be produced. The formation of an aneurysmal tumor gives rise to its own localizing symptoms. Resulting hemorrhage and thrombosis present symptoms related to the structures that are injured or destroyed.

The treatment of atheroma is practically the same as that of arteriosclerosis.

Arteriosclerosis, *arteriocapillary fibrosis*, is always a generalized systemic condition, but it may be more accentuated in certain bodily organs and there give rise to local symptom groups. Its effect upon cerebral activity is most important. According to Sansom, the changes brought about are due to a poison circulating in the blood, which acts upon the fibroid elements of various tissues, but preëminently upon those of the arterial channels. The essential histological modification consists of a fibroid proliferation or fibrosis. In certain locations this acts mechanically to strangulate associated structures, as, for instance, in the arteries, where the muscular fibers are thus invaded and even displaced. The walls of the entire arterial system become thickened. This may be due: (1) To thickening of the internal coat, which may go on to the complete obliteration of small vessels, or (2), commencing first in the external wall, the disease may spread inward, usually causing at first some hypertrophy of the muscular coat, or (3) the fibrosis may originate outside of the arterioles, which are involved secondarily by extension of the process to them. The various initial locations of the disease seem to depend upon the mode of the poisonous invasion. In one instance it affects the intima directly from the blood-stream, in another the outer coat from the perivascular or lymphatic space, and in the third variety the fibrous structures of parenchymatous organs are disturbed through the lymph-channels. All these varieties may be found in the same case.

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[1] K. Kompe, "Arch. f. Laryngol.," 1899.

This condition has a number of pathological associations. Atheroma is present in about one-half the cases. Cardiac hypertrophy and dilatation, hepatic cirrhosis, Bright's disease, asthma, angina pectoris, and mitral stenosis are frequently associated and due to identical changes. The effect of arteriosclerosis is to diminish the arterial caliber and thereby lessen nutrition. This may reach a complete degree and in the brain give rise to localized anemia and softening.

The nature of the poison which stimulates the fibroid activity is obscure. Loomis says the "general fibrosis has its origin in a fibroid diathesis either hereditary or acquired," but this explains nothing. By some writers defective elimination, particularly that from the kidney, is accused. Arteriosclerosis is certainly an accompaniment of old age and is a fair index of the wear and tear the individual has undergone and of the remaining vital capacity. We find it often a marked family characteristic. It is also clear that chronic intoxications by alcohol, lead, gout, rheumatism, and syphilis favor it strongly. Overeating, repeated muscular strains, and intestinal, renal, muscular, and cutaneous sluggishness count for something in its causation.

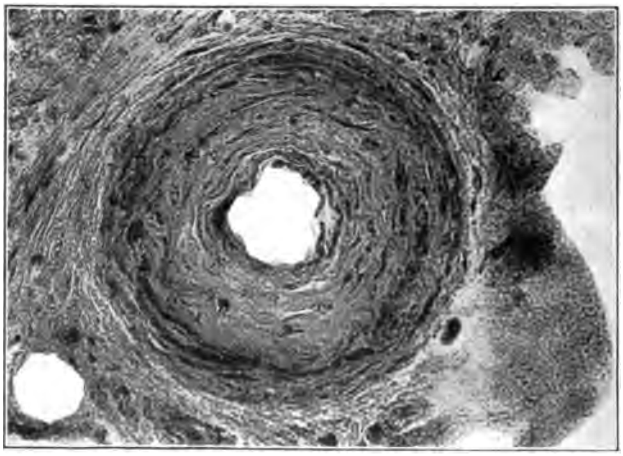

Fig. 84.—Arteritis: thickening of all the coats (Delafield).

The *cerebral symptoms* produced by arteriosclerosis cover a wide range, but are all due to faults of brain-nutrition. They embrace those of senility, premature senility, and degenerative processes, both chronic and acute. The highest and most delicate brain-functions are likely to be first affected. We, therefore, find lessened mentality, aphasia, and monoplegias or mere clumsiness of the hands. Paresthesias are very common. Hemiplegic and diplegic manifestations are observed. At first these symptoms are temporary and recurrent, but unless the arterial condition improves, they tend to become more and more continuous and permanent. Slight attacks of hebetude or sleepiness may eventuate in stupor, coma, and even in death. Jacksonian and generalized convulsions, syncopic attacks, and periods of mental confusion are all within the range of this protean malady. If the fibroid change occludes a cerebral vessel, it acts like a thrombus, to which, indeed, it often leads, and a softened infarct results in permanent loss of localized brain-function. Many islands of softening and many sclerotic patches may be due to this cause and present multiple symptoms. Diffuse sclerotic processes in the cortex are associated with it, and it underlies some of the cerebral lesions of general paresis and tabes. Associated minor symptoms, such as vertigo, headache, insomnia, irritability, lack of mental energy and muscular force, and the craving for stimulants, all point to the lowered nutrition of the brain. All the manifestations of cerebral arteriosclerosis are likely to come at first in gusts and waves.

Symptoms.—The physical examination of a case of arteriosclerosis usually demonstrates a tortuous, rigid, frontal artery, sometimes moving under the skin in a vermicular manner at each pulse-wave. The cornea commonly is the seat of a marked senile arcus. The radials, brachials, femorals, and all palpable arterial trunks are tense and rigid. The pulse is one of high tension. A light finger imperfectly detects it, but it seems to increase as pressure is applied and can hardly be obliterated. There is usually an enlarged, laboring heart, and often roughened valvular sounds. The urine is likely to show albumin and the formed elements that mark chronic nephritis. Often the quantity of urea is scanty or markedly deficient. A constipated habit is the rule, and the general health is below par. In less advanced cases the general indications of the arterial state may be very slight and yet the cerebral mischief may be extensive. This is especially true in the syphilitic varieties. The same processes that take place in cerebral structures affect the cord, and may, and often do, give rise to organic mischief, furnishing the basis of many systematized and unsystematized cord-lesions.

The treatment of arteriosclerosis affecting the brain must be undertaken at an early stage if much is to be accomplished. Toxic causes, if present, must be eliminated. Syphilis, gout, lead poisoning, alcoholism, renal, pulmonary, and cardiac conditions must receive their appropriate management. The arterial spasm due to the local irritation of the muscular tunic, and perhaps also to uric products in the circulation, must be overcome. To relieve the spasm the nitrites, especially nitroglycerin, may be given at short intervals. Mercurials and much drinking water to cleanse the intestines and stimulate the kidneys are valuable aids. Alkaline waters such as Vichy and the lithia waters are good. Care of the digestive tract and of the diet is of the first importance. All excesses must cease. A simple, easily digested regimen, with a very limited amount of red meats, starches, and sugars, should be ordered. The skin should be kept active by baths, frictions, and massage. If muscular exercises are for any reason contraindicated or not available, massage may take their place. An outdoor habit should be cultivated, and change of scene may do much to reduce the mental distress and vague broodings. Of all medicine directed toward improving the arterial condition the iodids easily hold first rank, but it should be remembered that the potassium salts depress the heart's action and perhaps add to the arterial sluggishness. Sodium iodid is much to be preferred and is usually better tolerated. This should be given in doses of from five to twenty grains after meals, and continued for months and years, with short intervals. A good plan is to order the iodid discontinued during every fifth week. Tonics are almost invariably required. Arsenic can be readily given with the iodid, and strychnin is perhaps the best aid to the laboring heart. Avoid digitalis and everything else that tends to increase arterial tension. No harm seems to come from the frequent use of amyl nitrite or trinitrin, and the prompt, though transient, relief produced is often very gratifying, besides in a way confirming the diagnosis.

Under such a plan of treatment, aphasias, mental disturbance, hemiparesis, and many other symptoms of brain disturbance will sometimes rapidly clear up and, if not cured, remain in abeyance for years, provided moderation in all things be the rule of life. The prognosis, however, should be guarded, as we know that brain-cells degenerate beyond recovery if entirely deprived of their blood-supply for about forty-eight or, at the most, seventy-two hours at a time. At best it is evident that the presence of arteriosclerosis signifies a shortened life-lease.

Syphilitic Arteritis.—It is now generally recognized that cerebral arteritis from syphilitic infection may be a comparatively early manifestation of the disease. Ogilvie shows from Naunyn's statistics that syphilitic diseases of the cerebrospinal axis present the greatest proportion of cases during the first year following the initial lesion, but they may appear even after a score of years. The basilar, carotids, circle of Willis, and large cerebral arteries are those usually implicated, but smaller brain-arteries may be similarly diseased. The specific inflammation may produce a peri-arteritis and nodular plaques that look something like those of atheroma, or it may invade and infiltrate all the arterial walls with gummy products, commencing either as an endarteritis or a peri-arteritis. Long-standing syphilitic arteritis, especially of the large vessels of the base, produces a sclerous degeneration that does not calcify. It is generally circumscribed in small patches, causing bulging of the internal and external coats, deforming the artery and altering its capacity.

Syphilitic arteritis leads to: (1) Obstruction of the vessel by the production of thrombosis or by an obliterating endarteritis; (2) rupture and hemorrhage, and (3) aneurysm. It may appear at any age and may follow inherited syphilis in children and even in adults.

The most prominent indications of syphilitic disease of the cerebral arteries are the prodromata. Of these the syphilitic headache, coming on usually toward evening and lasting until midnight, is the most distinctive. Except that due to tumor, it is the most intense and unmanageable headache with which the physician is called upon to deal. Ordinarily it is not confined to any portion of the head, but is described as being somewhat superficial, unlike the deep-seated pain of tumor. Unless the condition is now recognized or, as is rarely the case, spontaneously subsides, disturbances in the cortex are likely to appear, marked by paresthesias and loss of power in the extremities or disturbance of speech and the special senses. After vacillating symptoms of this character, which have a tendency to recur at intervals of a few days or weeks, an apoplectic stroke, due to sudden rupture of the vessel, may ensue, or a complete thrombosis lead to cerebral softening. When hemorrhage or thrombosis takes place the headache usually disappears, or sometimes it disappears a few days before the onset of serious results. The symptoms are those of cortical irritation and the eventual onset of paralysis is usually not marked by complete loss of consciousness, except in the hemorrhagic form, and coma is the great exception. Aphasia, facial paralysis, monoplegia, paresthetic tinglings, preceded by a history of violent headache, with nocturnal exacerbations, strongly indicate syphilitic disease of the cerebral arteries, even in the absence of any history of specific infection. As has been shown by Charcot, almost invariably there is some degree of basilar syphilitic meningitis in these cases and transient or permanent disturbance of the ocular apparatus is often added.

According to Charrier and Klippel, the chief groups of cerebral manifestations of syphilitic disease of the arteries are: (1) Apoplexy; (2) paralysis from obliterating arterial disease; (3) slight aphasia and transitory varying palsies, and (4) intellectual disturbance somewhat similar to that of general paresis. There is also reason to suppose that it is the true though remote basis of general paresis.

The treatment of the condition should be energetic even when it is diagnosed early, and will be discussed at length under the consideration of General Syphilitic Diseases of the Nervous System.

Acute arterial degenerations of an amyloid and fatty character affecting the cerebral vessels may follow numerous states marked by various systemic infections. Only in very rare instances do they give rise to marked cerebral symptoms, and these are usually overshadowed by the general state. Rupture and hemorrhage or thrombosis and softening may be due to them.

CHAPTER VI.

CEREBRAL HEMORRHAGE AND THE HEMIPLEGIC STATE.

EXCEPTING traumatic cases, hemorrhage into the substance of the brain is a secondary or terminal effect of degenerative or inflammatory disease of the cerebral blood-vessels, almost invariably of the arteries. Usually of comparatively slight seriousness in itself, the resulting injury or destruction of important brain-structures entails permanent disability if an early fatality is escaped. All parts of the encephalon are subject to arterial disease and resulting hemorrhage, but certain locations present special liabilities. The *most frequent site* is in the distribution of the lenticulostriate arteries arising directly from the trunk of the middle cerebral and supplying the basal ganglia and their internal and external capsules. Of these, the anterior branch is the so-called artery of cerebral hemorrhage. After the region of the basal ganglia and capsules, in order of frequency as sites of cerebral hemorrhage, follow the centrum ovale, cortex, cerebellum, pons, medulla, crura, and corpus callosum. So commonly, however, does hemorrhage occur in the neighborhood of the large ganglia that a somewhat definite clinical type of cerebral hemorrhage is presented. This will first engage our attention, and then the less frequent sorts and varieties, all of which have symptoms in common, may be rapidly sketched.

Pathological Anatomy.—Cerebral hemorrhage of the capsular variety occurs with about equal frequency on the two sides of the brain. The quantity of blood extravasated depends upon two factors, (1) the size of the blood-vessel and (2) the arterial pressure, but the pressure is much the more important of the two. According to Wernicke "the apoplectic or traumatic effect is equal to the product of the amount of effused blood into the square of the pressure with which it is extravasated." Ordinarily the hemorrhage commences in the anterior portion of the lenticular nucleus and separates or tears through the adjoining structures, invading the internal capsule, the external capsule, and the optic thalamus. It may extend upward into the centrum ovale or tear into the lateral ventricle, and finally break through into the pial spaces at the base near the optic chiasm. Very exceptionally the blood breaks through the cortex of the convexity, but ordinarily it is arrested at the lower surface of the gray matter.

Recent hemorrhages show a coagulum bathed in serum and non-adherent to the surface of the hemorrhagic pocket. After a few days the serum is relatively increased, the coagulum is contracted, and is attached to the adjoining structures by fibrinous trabeculæ. Absorption of the serum then follows. The clot is resorbed in part, and finally only an ocherous mass remains, made up of blood-crystals, pigment, and fatty detritus. By thickening and participation of the surrounding brain a sort of cyst is formed. If it is small and its walls coapt, cicatrization may ensue. About large so-called apoplectic cysts there is usually a zone of degeneration and fatty softening. This is often the seat of minute hemorrhages, and is likely to undergo inflammatory changes or by infection become purulent. An abscess thus results in which, sometimes, the partially organized hemorrhagic clot floats.

Even in recent hemorrhages there is difficulty in determining the origin of the blood. By carefully washing away the clot under water miliary aneurysms are almost certain to be found. They bead the arterial vessels that are brought to light, and sometimes one is fortunate enough to find the minute sac originally ruptured and still containing a fragment of organized clot. Other vascular lesions that occasionally give rise to cerebral hemorrhage are amyloid, hyaline, syphilitic, and fatty arterial degenerations and acute infective arteritis. In ancient cases cicatrization and spreading degenerative changes usually obscure the exact vascular fault.

The motor tracts that are cut by the lesion present a descending degeneration which extends downward the whole length of the neuron. In the cord the pyramidal tracts are usually both involved, but to a greater extent on the side opposite the lesion. Exceptionally in cases of long standing, changes in the upper motor tract entail secondary disturbance on the lower motor and trophic neuron, and an amyotrophic condition is superadded, with corresponding degeneration in the anterior horns.

Etiology.—In any given case of cerebral hemorrhage there are, ordinarily, a number of causative elements. Almost invariably there are (1) *high arterial tension* and (2) *lowered arterial resistance.* The causes of miliary aneurysm are much the same as those of rigid arteries, atheroma, and cardiac hypertrophy. The strongly acting heart drives the blood-column through the rigid aorta and carotid, and its full force falls upon the arteries arising from the circle of Willis. These are of comparatively small caliber, and, not having outlets by anastomosis, oppose a dead wall of resistance to the directly received cardiac impulse. Surrounded in turn by perivascular spaces and not supported by firm parenchymatous tissues, their walls weakened by age or infection and yielding at numerous points to the formation of saccular dilatations, arterial rupture naturally follows.

The predisposing causes of cerebral hemorrhage are those that produce or tend to produce the primal arterial disease. Advanced age, gout, alcoholism, rheumatism, plumbism, and syphilis are chronic states favoring it. *Acute infections,* tending to produce acute arteritis or amyloid and fatty degenerations, such as puerperal infection, diphtheria, scarlet fever, etc., are possible active factors. *Whooping-cough* in children is an occasional cause, the factors of strain, infection and lowered resistance all being present. An undoubted *hereditary tendency* to cerebral hemorrhage is sometimes encountered. The writer is familiar with one family of eleven brothers and sisters, nine of whom have died from cerebral apoplexy. The so-called apoplectic habit has no significance if not associated with other predisposing conditions. The relation of *age* to cerebral hemorrhage is an important one. During the first year of life it is relatively frequent, and drops thence to the end of the first decade. Gowers states that a proportion of 1.8 to every 1000 under the age of ten have suffered from this accident. From ten to twenty-five it is infrequent, and then rapidly mounts, reaching its maximum at about fifty-five, thence again descending, and seldom occurs after seventy-five years of age. The male *sex* shows considerable preponderance, about three males to two females suffering from cerebral hemorrhage. This is due to their greater tendency to excesses and increased liability to exposures. Temperate climates and winter months show an excess of cases over the opposite conditions.

Everything which induces an accelerated circulation must be ranked as an **inciting cause.** All physical, mental, and moral *shocks,* and all *intense emotions* may lead to cerebral apoplexy in those predisposed. Muscular *efforts,* coughing, sneezing, straining at stool, coitus, and vomiting have induced it. Derangements of the circulation with cardiac effort, due to exposure to the cold or from a cold bath or a bath after a full meal, have served to rupture the diseased artery. In the Great Lake region of this country the frequent sudden changes in barometric pressure, attended as they very often are by extraordinary variations in atmospheric humidity and temperature, constitute a menace, if they do not furnish an inciting cause of cerebral hemorrhage. It often follows a debauch.

Symptoms.—The *onset* of cerebral hemorrhage of the capsular variety is almost invariably abrupt, and constitutes the type of cerebral apoplexy that is familiarly and properly called "a stroke." In very exceptional cases it is preceded by momentary confusion, vertigo, uneasiness, or other vague subjective disturbance. Most of the alleged *prodromata* are merely symptomatic of the arterial condition that always precedes the stroke, and are common to peri-arteritis and arteriosclerosis. A hypertrophied heart, rigid artery, and high arterial tension, a contracted kidney and albuminuria, with or without formed kidney elements, are of some value in presaging an apoplectic seizure from cerebral hemorrhage. The patient, ordinarily, is stricken down, and in the foudroyant cases may die instantly. If walking, he falls heavily, or if sitting, reels out of his chair. Consciousness is almost instantly abolished. The action of any of the inciting causes named above will correspondingly affect the opening scene. At first the face is pale, the pupils contracted, and muscular twitchings or, rarely, active general convulsions may occur. There may be considerable motor restlessness and uneasiness until coma becomes profound.

In the **apoplectic state** that follows the patient lies inert, *unconscious*, breathing slowly and stertorously, and often presenting the Cheyne-Stokes respiration. The *face* is mottled or even deeply congested, covered with perspiration, and expressionless. The half-open *eyes* present sluggish or inactive and usually contracted pupils. The conjunctiva is usually congested and insensible, and the palpebral reflex is wanting. All forms of sensibility are abolished. The *sphincters* are relaxed and the limbs limp. There is usually urinary retention, and overflow occurs later. At this time it may be difficult to determine which hemisphere has been injured. Sometimes, by careful examination, the paralyzed side is found more completely relaxed than the other, in which, perhaps, some slight resistance to passive movements is present, or the sound limbs when raised do not fall so heavily. There is also, even from the first, a tendency for the patient to direct his face and eyes in *conjugate deviation* to the side of the brain that contains the hemorrhage. This indicates muscular paralysis on the side from which they are turned. This deviation must not be of the spasmodic sort, which has an opposite significance. At first all the cutaneous and muscle *reflexes* are temporarily increased, but immediately subside and tend to disappear. Sometimes they show most diminution on the paralyzed side. The toe-sign of Babinski almost invariably develops within a few hours, even within a few minutes, and persists indefinitely. The comatose condition either deepens into death or passes off.

After a variable period of hours or even days the coma, in cases not immediately fatal, gradually yields to torpor, in which the patient can be roused by strong and especially by painful stimulation. He pushes away the pinching fingers of the examiner, mutters a little, takes a deep, quick inspiration, or otherwise manifests discomfort. Then he hears loud voice-sounds, and finally shows, by facial expression and attempts at speech, that he has regained partial consciousness. If undisturbed, he relapses into stertorous sleep, and the expired air puffs out the flabby cheek of the paralyzed side of the face and escapes through the angle of the mouth on that side. Finally consciousness is restored.

Shortly after the stroke the rectal *temperature* is slightly lowered, but is likely to rise a little above normal after a day or two. In all cases after a few hours the temperature will be found higher on the side from which the eyes are averted—that is, on the paralyzed side—than on the other. This is true of the mouth, face, trunk, and extremities, and the difference amounts to from 1° to 2° F. At the same time this warmer, paralyzed side often shows increased perspiration, and usually an intensified redness and congestion as compared with the other.

The motor loss in this variety of cerebral hemorrhage is hemiplegic. After consciousness has partly returned its distribution can be plainly determined. Usually the arm is much more affected than the leg, and the face shows great variations of implication in different cases. In some it is scarcely affected. As a rule, the distal portion of the limb is much more affected than the portion near the trunk. The hand thus suffers proportionately more than the arm or shoulder, the foot than the thigh. The muscles of the trunk, being paired and fully represented bilaterally in the cortex, do not show much one-sided loss of power. A careful examination will, however, detect reduced respiratory excursions on the palsied side in forced respiratory movements, and unilateral weakness in the acts of sneezing, coughing, etc.[1] The *facial asymmetry* offers several important and significant peculiarities. Except in the rarest instances it is only the lower half of the facial-nerve distribution that is much impaired. The frontal and orbicular regions show but little lack of power; the brows are raised, the eyes close and open nearly as well as ever, but a slight unilateral paretic defect is certainly present as a rule. In the lower face the lack of muscular power and tone allows the mouth, chin,

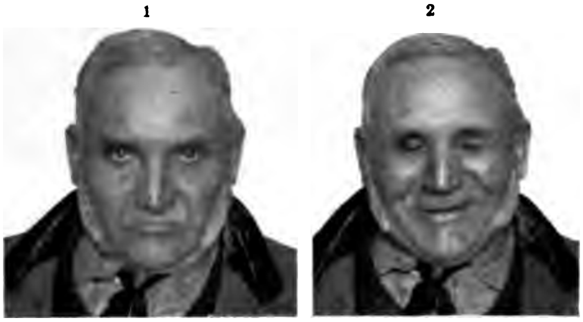

Fig. 85.—Right hemiparesis with athetosis in hand. 1, Ordinary expression; 2, spasmodic involuntary laughter, showing bilateral action of facial muscles.

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[1] Sicard, "Arch. de Neurolog.," Dec., 1899.

and even the nose to deviate to the sound side. All *voluntary* attempts to use the labial, nasal, and zygomatic muscles increase the deformity, but usually in *emotional* expression, as in laughing or weeping, the lack of symmetry tends to disappear (Fig. 85). Only when the basal ganglia, and particularly the thalamus, are seriously invaded by the hemorrhage do emotional expressions also intensify the one-sidedness. Even then the retention of power in the upper half of the face distinguishes this palsy from that of disease of the seventh nerve. The conjugate deviation of head and eyes passes away as consciousness returns, but the *tongue* when protruded turns strongly to the paralyzed side, owing to the weakness in the corresponding genioglossus. With the tongue protruded the patient can not move its tip across the median line toward the sound side. This lack of muscular power in the tongue and lips tends to muffle and thicken the *speech*, which may even become nasal, as the *soft palate* shares in the muscular weakening and droops on the paralyzed side. *Aphasia* is not ordinarily produced by capsular hemorrhage. In exceptional cases the lesion implicates the cortico-peduncular speech-tracts, producing a variety of motor aphasia marked largely by ataxic control of the muscles of speech-production.

Sensory Disturbances.—As consciousness returns after the stroke, ordinarily the complete cutaneous insensibility disappears. Only in those cases in which the lesion destroys the sensory pathway in the posterior third of the posterior limb of the capsule do we have persistent *hemianesthesia* corresponding to the hemiplegia. This identical anesthetic field is sometimes encountered in hysteria, which may indeed be induced by the shock of a cerebral hemorrhage as well as by any other accident, and may give rise to a perplexing combination. Close scrutiny for other evidence of antecedent hysteria should be made in all such cases. The sensory disturbance in the limbs is usually more pronounced toward their distal extremities, where the paralysis is also always most marked and persistent. *Hemianopsia* is frequently present immediately after the stroke, but usually passes away in a few days with the other sensory disturbances. When the visual path at the sensory crossway is injured, hemianopsia persists. Even when the patient is still inclined to stupor it may be detected by bringing the fingers into the visual field first from one and then from the opposite side. Disturbance of *other special senses* is subject to the same rule. Often there is considerable pain and sensitiveness in the paralyzed limbs. Severe headaches and vertigo are uncommon until the hemiplegic state is established, and even then they are rare.

Trophic disturbance in the early days after cerebral hemorrhage is rare. An *acute bedsore* sometimes appears over the buttock of the paralyzed side, and in a few days, almost in a few hours, attains large dimensions. Such cases almost invariably soon terminate in death. Rapid *emaciation* and even rapid *fattening* are encountered at times. Disturbance of the medullary nuclei probably accounts for temporary *albuminuria* and *glycosuria*, which are incidentally observed in some cases. To the same source some attribute the rare gastric and intestinal *hemorrhages*. Occasionally a large joint on the palsied side, notably the shoulder, may develop an *acute arthritis*, and all the articulations of the paralyzed limbs are later on inclined to rigidity out of proportion to their disuse. After the first week a rapid elevation of temperature is sometimes produced by the development of an infectious *cerebritis* about the hemorrhagic focus. It usually ends fatally.

The hemiplegic state finds its prototype in the cases that survive the stroke of cerebral hemorrhage. It must not be overlooked that it is a sequence common to many cerebral and spinal lesions, and is associated with some neuroses. It may follow: (1) Traumatic lesions of the brain and cord; (2) meningeal lesions due to hemorrhage, inflammation, syphilis, and tuberculosis; (3) cerebral lesions due to hemorrhage, softening, tumors, abscesses, and sclerosis; (4) cerebrospinal lesions of tabes, multiple sclerosis, and general paralysis of the insane. It may be due originally to: (1) Intoxication by uremia, diabetes, alcohol, lead, mercury, and some carbon compounds; (2) infections from pneumonia, malaria, typhoid fever, puerperal fever, eruptive fevers, diphtheria, influenza, syphilis, and tuberculosis; (3) it marks some cases of chorea, hysteria, and paralysis agitans.

In cerebral hemorrhage, if death does not occur during the first three weeks, improvement in the paralytic features uniformly appears and is progressive for several months. Finally, all gradations are encountered, from the slightest paresis to complete hemiparalysis. In a well-marked case, in which at first the hemiplegia is complete, we may expect some return of voluntary motion after ten or fifteen days. The lower extremity first shows improvement, and is followed by the face and later by the upper extremity. In six to eight weeks the patient may stand and perhaps with much aid walk a little. During this time a notable change has appeared in the reflexes and the tone of the paralyzed muscles is greatly altered. At first the reflexes are abolished or greatly diminished and the muscles hang limp and flabby from the bones. Gradually the reflexes increase and become exaggerated. Transient stiffness and rigidity are then found in the limbs. Ankle-clonus, rectus-clonus, and even wrist-clonus appear, and with the temporary rigidities foreshadow the later contractures. The increase in myotatic irritability may be expected to appear during the third week, and is always present sooner or later, if the motor pathway in the brain is injured by the hemorrhage.

Introduced by the myotatic irritability, clonus, and the fleeting attacks of rigidity, the *period of late contractures* is established from one to four months after the stroke. In very rare cases it is never fully developed, and in other rare cases, owing to secondary changes in the trophic centers of the cord, it disappears after being well marked for years and muscular atrophy develops, but the rule is that once present it is progressive for a time and then permanently remains. Under the action of this contracture the limbs assume characteristic rigid attitudes that constitute great deformities. It sometimes strongly involves the *face*, which is then drawn to the affected side and gives to the sound side a false appearance of weakness.

In the *upper extremity* the flexors predominate to draw the digits into the palm of the hand, to flex the wrist, to pronate the forearm, and to fix the elbow at an angle. The extremity is usually held close to the chest. In these vicious positions the *joints* become practically soldered. After the case is somewhat chronic, if the fingers or wrist are extended, they fairly snap back to their flexed positions, and the tightly clenched fingers may cause trophic lesions in the palm. In cases of long standing, probably both from the enforced inactivity and trophic disturbance, the range of joint movements is considerably limited irrespective of the muscular contracture, and sometimes this appears early.

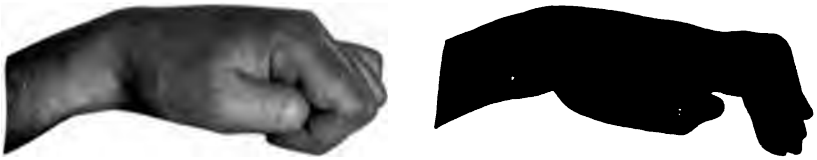

Fig. 86.—Contractured positions in hemiplegic hand.

In the *lower extremity* extension prevails. The knee is held quite rigidly extended and there is a tendency to equinovarus, so that the foot rolls over on its outer border. These are the usual contractures, and give to the patient an *attitude* and a *gait* that are distinctive. It will be recalled that the distal portions of the extremities are most affected, and

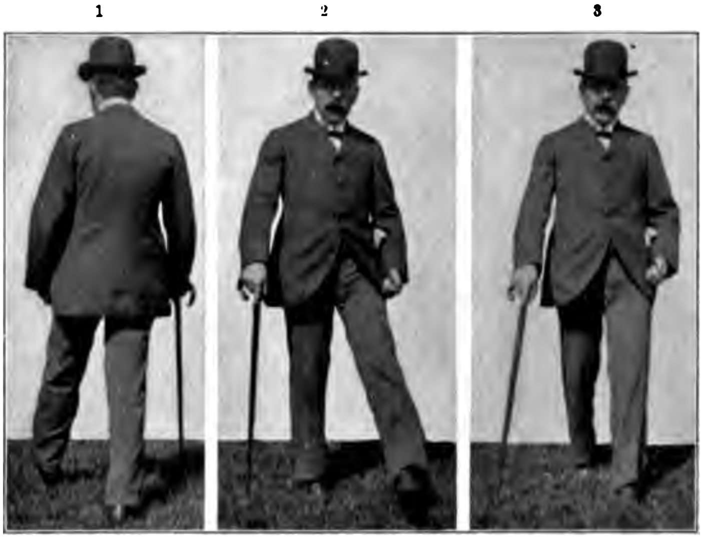

Fig. 87.—The hemiplegic gait. 1 and 2, Advancing the hemiplegic leg in a circle from the hip; 3, bearing weight on paretic leg and cane while advancing sound limb.

now, rigidly fixed by the contractures, they are moved *en masse* by the muscles of the pelvic and shoulder girdles. In *walking* the rigid lower extremity is used somewhat as a peg-leg. The body-weight is carried on the sound limb, over which the trunk inclines outwardly, and by body and pelvic movements the paretic lower limb is swung forward, the dragging toe describing an arc-like course around the heel of the sound side.

Then upon the paralyzed limb, often trembling with clonic action, as upon a stilt, the next step is taken by the sound side, aided usually by a cane or crutch.

A marked minority of cases show a type of extension in the upper extremity, which is most pronounced at the elbow. The limb is held rigidly by the side of the body. The wrist and fingers may, though very rarely, also be extended, and the supinators may overbalance the pronators. Again, flexion may predominate in the lower limb, or extension may be present in the upper and flexion in the lower limb, or different segments of the same limb may show opposite conditions as to flexion and extension. This late rigidity may render it impossible to demonstrate the *muscle reflexes*, which, nevertheless, are greatly exaggerated. The toe-sign is usually present and readily elicited. Like the myotatic irritability, the late contractures are due to the descending degenerative processes in the motor tracts.

The paralyzed limbs show marked *circulatory disturbance*. They are at first reddened and cyanosed. The blanched spot caused by finger-pressure only slowly disappears. Sometimes they are sodden with perspiration and show epithelial variations, either by increase or more often by decrease of growth in the nails, hairs, and epidermis. They are cold later on, and their temperature is reduced several degrees as compared with the sound side. At times there is marked edema, which may come on very early in the case, and is attributable to the muscular activity, lymph-stasis, and frequently to a kidney or cardiac lesion.

A phenomenon is often noticed by hemiplegics that gives rise to false hope of returning voluntary movement in the paralyzed limbs. Sometimes without volition the paralytic hand may open or the leg draw up. These movements are perhaps analogous to the constant *athetoid features* that develop in some cases. The entire upper extremity, over which the patient has practically no control, may execute such movements as raising itself over the head in conjunction with the other arm in yawning, stretching, or other instinctive act. These *associated movements* probably result from the bilaterality of their cortical representation. Again, if the hemiplegic attempts to write with the sound hand, provided it is the one not formerly used for that purpose, the paralyzed hand may sometimes be seen to duplicate the motions of the one voluntarily used. Right-sided hemiplegics who are at the same time right-handed, in attempting to write with the left hand sometimes produce *mirror writing*, which is practically the normal method for left-handed individuals.

On the sound side there are also marked disturbances. The reflexes are exaggerated and even contracture may develop. The muscular strength is reduced from 10 to 50 per cent. Incoördination may be pronounced.

Complications.—The hemiplegic state often presents *sensory complications*. Subjectively these consist in paresthesias, such as feelings of heaviness, dragging, and formication. Sometimes the limbs seem to the patient to be entirely gone; others complain of phantom or additional limbs. Objectively we may have hemianesthesia if the sensory portion of the capsule is injured, and the other sensory disturbances of injury to the great crossway may be added.

A wide range of *posthemiplegic motor complications* are encountered. We find rhythmical tremors, as in hemiparalysis agitans, or the intentional variety of tremor, as in multiple sclerosis of limited distribution. Irregular movements, like those of chorea, ataxia, and athetosis, are quite common on the affected side. When they result from bilateral cerebral lesions, great diagnostic difficulties are presented. The production of all these posthemiplegic motor symptoms requires that the paralysis should not be complete, and their presence implies a condition of irritation somewhere in the path of the upper motor neuron. This is usually furnished by lesions in the region of the basal ganglia, especially those affecting the optic thalamus and impinging upon the capsular fibers.

Among the *trophic complications*, in addition to the early, acute bedsore, the paralyzed side may present indolent ulcerations over the sacrum, elbow, or heel. The *amyotrophic* disturbance already mentioned may come on within a few days of the stroke, or may appear late in the hemiplegic history, and, in either event, must be attributed to interference with the trophic control of the anterior horns. The electrical reactions then vary from those of simple quantitative changes to the reaction of degeneration. This muscular wasting is most common in the upper extremity, and, when appearing early in the case, is of ominous import. The skin, hair, nails, and fatty dermal layers may be increased or more rarely diminished in the paralyzed limbs.

It would also seem that dynamic, if not actual, changes in the anterior horns must be accountable for the occasional *acute hypertrophic neuritis* that develops in the paralyzed limbs in the early days after the illness, and for the acute *arthropathic affections* that also appear at that time. These are sometimes associated. The diseased joints suggest rheumatism. They are inflamed, painful, hot. The joint-surfaces and synovial organs rapidly disintegrate, and present practically the same condition as the arthropathies of tabes. When appearing early this joint affection is of bad import, like the acute decubitus and early amyotrophy.

Hemiplegia from the ordinary capsular hemorrhage does not necessarily disturb the *mind*. In the old it may precipitate the mental deterioration of senility, and it may be followed by organic dementia.

The course of cerebral hemorrhage of the common type may present many variations. Death may occur almost instantly, but this is exceptional, and usually the result of inundation of the ventricles. Death may occur during the coma from the great size of the clot and the exhaustion of the patient. After coma has disappeared and before contracture has developed, death may result from cerebritis arising from infection of the clot. During this early period pneumonia, often caused by the aspiration of food or drink, sometimes carries off the patient. A succession of hemorrhages taking place at short intervals, one apoplectic phase succeeding another, are nearly always fatal. After the initial stroke, and usually before the termination of the coma, there may be a sudden increase in all the symptoms. The breathing becomes rapid, irregular, stertorous, the unconsciousness more profound. The reflexes, if partly present, are entirely lost, and tetanic spasms or convulsions appear on the paralyzed side. The temperature bounds upward and the case terminates fatally in a few hours, or in a day or two. These symptoms are due to the hemorrhage bursting into the ventricles. If the fourth ventricle is invaded, nystagmus appears and death promptly follows. In the so-called *ingravescent apoplexy* the hemorrhage begins without marked stroke, and, steadily continuing, produces the apoplectic state in the course of a day or two, finally inundating the ventricles and terminating fatally.

Recoveries from cerebral hemorrhage are not rare, and in the majority of cases the hemiplegic state is reached and the stage of contractures is developed. Very exceptionally the hemiplegia practically disappears. This is only possible when the hemorrhage has affected the capsular tracts by pressure without producing rupture of their fibers, and then the clot is necessarily small. As the greatest rarity the recovery may be so complete that no trace of the former palsy can be discovered by clinical examination.

Clinical Forms.—Cerebral hemorrhage presents a number of clinical forms, the most usual of which has been the basis of the preceding description. Variations in localization and in the amount of extravasated blood endlessly modify the type. Within the field of the lenticulostriate arteries a very small hemorrhage may produce a partial hemiplegia or a monoplegia, though this is a rare form. We have the occasional form of hemiplegia with hemichorea, or hemiataxia, or hemiathetosis. There is the form of hemiplegia with hemianesthesia and another in which hemichorea and hemianesthesia are combined. A rare form consists of a facial monoplegia with hemianesthesia, and a form presenting hemianesthesia and motor aphasia has been noted. Hemorrhage into the posterior lobes of the brain is attended by the apoplectic onset or insult and leaves visual or aphasic remnants if it involves the corresponding radiations and pathways. In the anterior lobes and in a large portion of the centrum ovale a considerable hemorrhage may take place without producing lasting symptoms. In these hemorrhages remote from the capsule hemiplegic symptoms are sometimes present at first and then disappear as the traumatic effect of the apoplexy subsides. Primary cortical hemorrhage is exceptionally encountered. The clot is usually small, owing to the small caliber of the cortical arteries; the stroke, therefore, is slight or absent, but stupor and semicoma may be present. The symptoms produced are those of an irritant lesion. If it occurs in the motor zone, repeated limited convulsions of a Jacksonian type are likely to occur and may become generalized. Hemorrhages into the pons and medulla, if not immediately fatal, give rise to localizing symptoms and cranial-nerve disorders that have already been discussed, including the numerous crossed paralyses.

Cerebellar hemorrhage usually presents a very sudden onset, but there is correspondingly much less disturbance of the mind and consciousness than occurs in the cerebral variety. The patient falls, vomits, and experiences intense vertigo. Attempts to sit or stand greatly increase the vertigo and vomiting and may render the horizontal attitude obligatory. If the lesion approaches the middle peduncle, forced movements or rigid positions may be induced. These may impel the patient strongly to the right or left, backward or forward, and in the reclining posture cause marked curving of the trunk in corresponding directions. Ataxia and asthenia in the extremities are immediately produced, and tetanoid spasms may appear. Sensory disturbances are fleeting or absent. Later on the preponderance of symptoms is on the side of the lesion if it is unilateral, but if pressure occurs on the pyramidal tracts above the decussation, crossed symptoms are presented. Cranial-nerve impairment is likely to be present from irritation of the nuclei and pressure on the floor of the fourth ventricle may induce dangerous pneumogastric complications. Should this ventricle be flooded, bulbar symptoms are induced and death promptly follows. Much similarity is presented by attacks of labyrinthian vertigo, and at first the diagnosis may be impossible. A history of preceding attacks and of ear disease is significant. If the cerebellar hemorrhage involves the auditory nerve, the two symptom groups coincide.

The diagnosis of cerebral hemorrhage is often difficult and sometimes impossible. Its consideration falls into two parts: (1) The diagnosis during the apoplectic state and (2) the diagnosis after the apoplectic state.

Shortly after the onset of the stroke we have to ask ourselves whether the case is one of *syncope*, *poisoning*, or *alcoholic intoxication*. If one-sided symptoms can be detected, all of these conditions may be excluded, and each has some distinctive symptom that one on his guard may detect. The pale face, fluttering pulse, and sighing respiration of syncope, the contracted pupil of opium, the smell of alcohol on the breath, etc., are suggestive, and taken with the history of the onset, when obtainable, are nearly sufficient. But cerebral hemorrhage may come on during drunkenness, or a hemiplegic case may be given stimulants. *Uremia* is more difficult to differentiate, and may occasionally counterfeit all the indications of cerebral apoplexy. Examination of the urine may throw light on the situation, but it is to be remembered that the conditions giving rise to uremia are those usually associated with arterial disease in the brain. Most uremic comatose cases, at some period, present vomiting, headache, motor excitement and spasmodic restlessness, in which paralytic features are lacking. The coma is rarely profound at first. *Meningeal hemorrhage* may be easily mistaken for cerebral hemorrhage, as stupor, coma, convulsions, and paralysis are produced by both. The meningeal form, however, usually follows injury, and comes on slowly or after a distinct interval. It is often marked by irritation of cranial nerves and early bilateral convulsions. The *epileptic attack* usually has a history of anterior convulsions, presents clonic, tonic, and stuporous stages, and is quickly and completely recovered from. Attacks of *focal epilepsy* may indicate a cortical hemorrhage, but are common to all diseased conditions of the cerebral motor surface. Every case must be carefully analyzed, and often, even then, only a presumptive diagnosis can be finally reached. *Hysteria* in rare cases produces an imitation of cerebral hemorrhage that is extremely faithful to the type. The attack, however, usually occurs under circumstances of emotional and psychical disturbances indicative of hysteria. The face is rarely involved, Babinski's toe-sign is absent, and sensory stigmata are commonly present. The age and clinical history are also significant. In *general paresis* apoplectiform seizures are common, and the differential diagnosis during the attack may be very difficult. A history of mental confusion, childishness of conduct, forgetfulness, unsteadiness upon the feet, and syphilitic infection would favor the paretic side of the question. Almost complete recovery from the stroke may then be expected in a majority of cases.

After the early apoplectic symptoms have persisted many hours or have subsided, an organic lesion can usually be determined from the hemiparalytic features. The important question regards the nature of the vascular accident. Is it rupture or occlusion that has taken place? Have we to do with hemorrhage or with infarction? Are we in the presence of extravasated blood with a tendency to encapsulation, or confronted by thrombosis, the precursor of softening? It is a question of importance, both as to treatment and prognosis. It is sometimes an unsolvable problem, but should never be neglected. At the end of the next chapter a differential table is given, to which attention is now directed. In a general way we may say that the following points favor a diagnosis of cerebral hemorrhage: Sudden onset, absence of syphilis, and endocarditis, the presence of strong cardiac action comparatively early, high arterial tension, marked coma and cyanosis, lowered rectal temperature and raised unilateral temperature, convulsions involving the whole of one side, paralysis involving the whole of one side, early improvement in the paralysis most marked in the leg, lack of permanent sensory disturbances, the presence of complete hemianesthesia in the absence of hysteria, the development of postapoplectic tremor, and athetosis.

The topical diagnosis must follow the general considerations laid down in the discussion of the subject of cerebral localization. Attention is again called to the fact that the above description is dominated by the clinical aspects of the usual capsular variety of cerebral hemorrhage.

Prognosis.—A cerebral hemorrhage is always of serious import, as it is a conclusive demonstration of wide-spread and threatening arterial disease in the encephalon. However slight, it implies the probability of a recurrence. About two-thirds of all the cases survive the first attack, one-third the second, and very few the third. The size and location of the clot are important considerations. The prognosis is grave in proportion to the violence of the attack and the depth and duration of the coma. Coma lasting three days seldom ends in recovery. The appearance of Cheyne-Stokes respiration or indications of ventricular flooding practically mean death. The occurrence of convulsions is a serious feature. Pneumonia is almost invariably fatal. Acute bedsore and acute joint disease are usually followed by death. A recurrence of coma or a sudden elevation of temperature or repeated apoplectic features imply an early fatality.

Unless there is some improvement in the paralysis at the end of the second week, it is likely to be permanent. The appearance of contractures implies lasting functional loss wherever they develop. The state of contracture commences from twenty days to three or four months after the stroke. The shoulder and hip movements improve more than the knee and elbow; the ankle and wrist, toes and fingers, progressively decline in recuperative prospects. The lower extremity surpasses the upper both in point of time and extent of recovery. Considerable improvement may be anticipated during the first two or three months, and then much slighter progress to the end of the first year or eighteen months. Thereafter the case will be practically stationary. The duration of life among apoplectics is about five years, according to Dana, and, as he points out, a stroke may be a conservative measure, enforcing an inactivity that prevents arterial strain and thereby prolongs life.

Treatment.—If a positive diagnosis of cerebral hemorrhage is made, immediate active treatment should be instituted, but in doubtful cases a masterly inactivity—an armed neutrality—is the proper course; there are many who think it the only course. The important indication is to *reduce arterial tension*, to discount the pressure at the site of hemorrhage. When the case is seen immediately after the stroke the head should be raised, an ice-cap applied, and faithful carotid compression on both sides of the neck employed for forty minutes. Meanwhile the blood should be directed to the lower extremities by having them swathed in hot compresses and by the intelligent application of mustard. Three drops of emulsified croton oil serve to practically bleed the patient into his abdominal vessels and at the same time unload the bowels. Regarding *venesection*, the forces are still divided. In plethoric, congested, livid cases with strong cardiac action it may properly be used. Aconite and veratrum are by some accounted equally valuable to bleeding, but they must be given in frequently repeated, competent doses. For instance, aconitia, $\frac{1}{200}$ of a gr., or fresh Norwood's tincture of veratrum, two drops every twenty minutes until effects are produced. Bromids and ergot are only mentioned to be condemned. In some cases a failing heart requires active stimulation, and alcohol and strychnin best serve the purpose.

In most cases the hemorrhage reaches its maximum within three or four hours and the damage is done. Thereafter the indication for treatment is solely to *prevent a recurrence*, and that means to keep the arterial tension down. Quiet, warmth, liquid diet, if any, free bowels, cleanliness to prevent bedsore, the administration of cardiac sedatives; attention to the bladder, which is likely to distend and overflow; care to prevent aspiration pneumonia, from food, mucus, or a septic condition of the mouth, and readiness to meet recurrent hemorrhages or ingravescent apoplexy by carotid pressure will answer the major requirement. Ligation of the carotid is not a well-established proceeding, and carotid compression must be used with circumspection and intelligence. In the aged and atheromatous it may produce convulsions or serve to increase the coma. The effect in controlling the circulation may be observed in the usually prominent and throbbing temporals and in the color of the face. Suggestions of early trephining and evacuating the clot have been made. If rupture of the motor path occurs directly the extravasation of blood takes place, further surgical laceration can scarcely improve matters. If purulent cerebritis develop in the clot, trephining to drain such a focus may reasonably be attempted in this otherwise fatal complication.

As soon as the apoplectic coma has passed away, gentle massage of the paralyzed side and exercise of all the paralyzed muscles by mild faradism should be instituted. The purpose should be clearly in mind to secure as much improvement during the first month as possible and to postpone to the utmost the appearance of contracture. A muscle that may feebly respond to volitional control, perhaps to the associated action of the sound hemisphere, is rendered perfectly useless by contracture. From the onset extremely gentle passive movements of all the joints to their full range should be employed every two or three hours. Every one is familiar with the rapidity with which muscular and joint stiffness appears in splinted extremities. In these hemiplegic cases the limbs are splinted by paralysis. As soon as there is any reappearance of voluntary motion the patient should be encouraged to exercise it. These measures are usually postponed to the end of ten days or a fortnight in fear that, by instituting them early, the brain-lesion may in some way be increased. Certainly any violent or severe measure is most strongly to be deprecated, but a common-sense application of the foregoing directions will be found to yield encouraging results.

When contractures appear, every effort should still be persistently made to counteract their deforming effect. We are familiar with the vicious attitudes they produce. In the upper extremity the extensors should be encouraged by massage, electricity, and forced passive movements to overcome the usual flexor supremacy. Similarly, in the lower extremity the equinovarus should be overcome, if possible, by stimulating the antero-external muscles of the leg. Rigidity at the knee is less objectionable. Every additional week of suppleness may mean increased voluntary control. Now, also, the patient should be constantly encouraged to concentrate his attention upon the paralyzed side and repeatedly attempt to move the paralyzed muscles, aiding the effort by the simultaneous use of the sound side in executing the wished-for movements. Weakness of the sound side may defeat efforts at walking, unless the patient is greatly encouraged and strongly supported. The distrust of their strength and preference for inactivity must sometimes be actively met by the encouragement and authority of the physician. After two years no further gain is to be expected, but even then locomotion may be facilitated by orthopedic apparatus or by a tenotomy of the heel-tendon. There are very few hemiplegics who escape more or less persistent medication with *iodid*. Aside from syphilitic cases it can not be strongly urged. As a treatment of the basic arterial state, however, it often has a legitimate place, and therein may be prophylactic of a second attack of hemorrhage. Too often it seems to degrade the patient's general health without corresponding benefit. It should go without saying that general hygienic and tonic measures are always indicated. The enforced inactivity of the hemiplegic favors intestinal atony and cutaneous sluggishness, which require watchful and intelligent management.

CHAPTER VII.

CEREBRAL SOFTENING.

THE term "cerebral softening" is open to objection for several reasons, but usage has confirmed its rank. "*Softening of the brain*" is a lay expression usually applied to dementia, and based upon somewhat erroneous notions of the condition of the brain in such cases. By *cerebral softening* is here meant the retrograde process in vascular territories of the brain, occasioned by arterial obliteration and local deprivation of blood-supply. It is an *infarction process* exactly similar to that which is common in the spleen and kidney. The brain-structure, however is not of a uniform functional value, and a cerebral infarct in the majority of instances destroys specific brain-powers instead of simply reducing the capacity of the whole, as in the abdominal organs mentioned. In addition it gives rise to secondary conditions similar to those following cerebral hemorrhage. It is to Virchow that we owe our first exact knowledge of the genesis of cerebral softening. Previously the softened atrophic area was attributed to inflammation or some vague morbid process. We know now that the immediate cause of the *encephalomalacia* is the occlusion of the cerebral vessel that supplied the diseased portion. This obliteration of the lumen of the artery arises (1) from thrombotic obstruction developing on the site, or (2) from plugging by an embolus starting at a distance, or (3) from local thrombosis following the lodgment of irritant emboli.

When a cerebral artery is occluded, the blood-supply of a tolerably definite territory is cut off because of the anastomotic defects of the cerebral circulation. In the case of a sizable vessel the center of its arterial field is wholly deprived of blood, but the margin is only relatively impoverished, as there is some overlapping of these vascular territories, by slight anastomoses and in some cases by direct anastomotic relations with the veins. The moment an artery is closed its distal portion is deprived not only of blood, but of blood-pressure, and, aided by its own resiliency, it tends to collapse. This gives opportunity for back pressure from the return or venous circulation, and the territory deprived of arterial inflow may become the seat of venous congestion. Moreover, the arterioles deprived of their nutrient contents are disposed to promptly degenerate, and thus arise the punctate hemorrhages so commonly found in comparatively recent cases of cerebral thrombosis. Subsequently, retrogressive changes take place looking to the removal of the mortifying focus. Fatty degeneration and phagocytosis go on rapidly. The coloring-matter of the extravasated blood finally alone remains. Repair takes place about the focus of disease, and it becomes encapsulated. In some infarcts of minor size cicatrization ensues, and a scar is left to mark the location of the vascular lesion.

Pathological Anatomy.—The first effect of arterial occlusion is to cause anemia in the distal portion, and the territory of distribution becomes blanched. The tissues degenerate, necrosis follows, and *white softening* is produced. If, from the return circulation and local punctate hemorrhages the strangled area becomes suffused and infiltrated with blood, color is added and *red softening* is presented. Finally, as resorption takes place and degeneration becomes complete a yellow color from the remnants of the hemic pigment marks the softened and perhaps encapsulated tissue, giving rise to the name *yellow softening*. The three appearances are but stages of the same process, but *white softening* is not necessarily followed by the red and yellow changes. The red appearance, due solely to the hemorrhagic element, appears, if it appears at all, after a few hours or a few days, depending upon the activity of the venous back pressure and the degeneration of the arterioles.

In a very few days after arterial obstruction has occurred the corresponding cerebral tissue presents a marked appearance of degeneration. It is a fact of practical bearing that nerve-cells, deprived of their nutritive supply for about forty-eight hours, are permanently ruined. The softening focus is infiltrated with serosity and the cellular and neuroglial elements are already breaking up. The myelin separates into droplets and is rapidly taken up by migratory leukocytes, which attain large proportions and have by some been described as granular bodies. The degenerating focus shrinks and softens. If situated beneath the pia, the resulting depression is filled with a turbid, milky fluid, and the soft meninges are thickened and highly vascular. It becomes impossible to strip the pia from the gyri of the softened area without decorticating them. The cortex is pale and friable, the white substance softened often to diffluence. In old foci of softening the surrounding tissue is thickened, especially in its neuroglial makeup, and presents an indurated wall within which a yellowish fluid substance containing fat-crystals and amorphous matter represents the former cerebral structure. Yellow softening may be found at the end of six weeks, but is a feature of old lesions. These may even become calcareous. *Cicatrization*—the fibrous obliteration of small softenings—has been already mentioned. In some cases the softened focus becomes infected, as by an embolus from infectious endocarditis, or from pneumonia, or from any infection atrium, and a secondary true *encephalitis* is developed. This rapidly goes on to *abscess* formation, frequently with putrid, offensive, gangrenous contents. Thrombosis occurring in the perforating arteries of the base, which are entirely without anastomoses, or in the deep cortical branches which penetrate the centrum ovale, produces rounded islets of necrosis, which in time may become encapsulated and contain only serous fluid.

At the seat of arterial obliteration arising from local thrombosis we find, in recent cases, a partially or completely organized clot adherent to a spot or ring of endarteritis or atheromatous thickening. An embolus does not at first present adhesions to the intima. It may be made up of pure fibrin from the endocardium, or calcareous particles originating in cardiac or arterial atheroma. Hydatids, filaria, and any minute substance finding access to the blood-stream may furnish a cerebral embolus. In about nine cases out of ten it originates in or near the heart, and in fortunate cases the exact location of its origin has been detected. Ordinarily an embolus lodges at the branching of an artery, from which point the thrombotic fibrin deposit extends. If the embolic mass be calcareous, it may abrade the intima and give rise to a dissecting aneurysm, which in turn leads to occlusion and thrombosis. The size of the softening depends entirely upon the size and relations of the occluded vessel.

The *location of cerebral softening* is more frequent on the left than on the right side of the brain. The left middle cerebral is especially selected, and its perforating or basilar are more frequently involved than its cortical branches. This is due perhaps to the fact that cardiac emboli following the direct route tend to enter the vessels that most conform to the straight line of their momentum. These are also the favorite seats of atheroma. The anterior capsuloganglia region is the most common site of cerebral softening, followed in decreasing frequency by the posterior capsuloganglia region, the cortical territory of the middle cerebral, that of the posterior cerebral, then that of the anterior cerebral. Softening is rare in the cerebellum and in the bulb, except in syphilitic endarteritis, which frequently implicates the brain-stem. In rare cases we encounter a bilateral and symmetrical softening. In some instances this may be explained by thrombosis or embolism starting in the circle of Willis. For instance, an atheromatous patch at the bifurcation of the basilar may furnish a plug to the left side, and circulation on that side being proportionately reduced, the next embolus goes to the right side. In some cases there are numerous foci of softening. Softening may affect only the gray cortex, or the white subcortical substance, or both. The extent of necrosis depends always on the damaged arterial supply. If the Sylvian trunk be occluded, the entire sensorimotor zone is softened as well as the subcortical white substance as deep down as the basal ganglia. If the thrombosis or arterial stagnation implicates only the short cortical branches, local death of the brain-mantle alone may follow. Again, if the arterial disturbance affects the long penetrating arteries that traverse the cortex to supply the centrum ovale, the cortex may be spared.

Etiology.—The ultimate causes of cerebral softening are those of the arterial diseases, already considered, that underlie thrombosis and embolism. In a rough way we may say that thrombosis, usually a sequence of atheroma and arteriosclerosis, pertains to advanced age, alcohol, lead, gout, and syphilis, and that embolism is an accident of left endocarditis due to acute rheumatism and the infections of pneumonia, typhoid, diphtheria, the puerperium, etc. As a presumptive rule, we may also say that cortical softenings are commonly due to embolism and occur most frequently in youth; that those of the central area are due to thrombosis and take place in advanced years. Thrombosis is favored by a weak heart and sluggish arterial current of low tension, conditions ordinarily found in cachectic and marantic patients and favored by sleep. Embolism is favored by any act that throws a load upon the heart and produces cardiac stimulation. Vigorous muscular efforts, sudden emotion, or merely rising to the feet from a reclining position may start an embolus when friable arterial or cardiac vegetations exist. Very exceptionally a cerebral artery is obliterated by conditions arising outside its lumen, as by a rapidly growing tumor or other compression factor acting with some degree of promptness. Unless the compression is brought rapidly to bear, the circulation is able to adjust itself. Heredity, sex, age, temperature, barometric pressure, and the seasons are of much less significance than in cerebral hemorrhage.

Symptoms.—The early symptoms of cerebral softening are dominated by the rapidity with which the arterial current is blocked and by the importance and size of the vessel involved. As a matter of fact, embolism is capable of producing instantaneous blocking of the arterial lumen, and symptoms of great violence at the onset to some degree indicate embolism if hemorrhage is excluded. On the other hand, while thrombosis usually produces progressive symptoms, a slowly growing thrombic arterial lining may cause a sudden deposit of fibrin from the blood, and intense symptoms are thereby precipitated. Again, a small embolus may only partially choke the blood-current, and the slowly developing occlusion is marked by the deliberately advancing symptoms and prodromata of the stroke. Cerebral thrombosis, therefore, presents both (1) an abrupt and (2) a progressive onset, with different cerebral symptoms immediately resulting, but with the same terminal conditions.

The Abrupt Onset.—With none or only momentary confusion the patient has a stroke or cerebral attack, followed by loss of consciousness, perhaps even by the development of coma. Hemiplegia develops, consciousness returns, and more or less paralysis persists. In about an equal number of cases consciousness is not lost, though the paralysis is as quickly developed, and, as a rule, the mental subjugation, the cerebral insult, is less than in hemorrhage. Many cases are attacked during sleep, and awake at their usual hour with no appearance of stupor and with their ordinary mental brightness. A very significant feature of thrombosis affecting the cortical circulation is the Jacksonian or limited convulsions which are commonly presented, and which tend to repetition during the early hours and days of the illness without great, often without any, disturbance of consciousness. Distinguishing softening from hemorrhage, we seldom encounter the retarded strong pulse, the subnormal temperature, the unilateral heat, the congested face, the respiratory difficulty, and the prolonged coma so characteristic of a ruptured cerebral vessel. In a fair proportion of all cases of thrombosis the early symptoms are progressive. A monoplegia becomes a hemiplegia. Paralysis beginning in the leg invades the entire extremity, reaches the upper limb, implicates the face, disturbs speech if on the left side of the brain, and finally the hemiplegia is complete. This is due to the thrombus, by gradual increase, backing down the artery from its original place of development, and if it extend into the circle of Willis the opposite Sylvian artery may also be blocked. When the basilar is invaded bulbar symptoms usher in a fatal termination. Thrombosis beginning in the branches of the middle cerebral may first produce an aphasia; lower facial paralysis and loss of power in the limbs of the same side may then be gradually added. The distribution of the arteries to the motor region makes clear the sequence of events. (See page 194.)

The Progressive Onset.—When the arterial obliteration is a slow process and the caliber of the vessel is gradually reduced, disturbances in the poorly irrigated cerebral district are likely to be manifested. In the case of a Sylvian vessel this is the rule. The patient complains of more or less transient or persistent sensations of fullness, heaviness, formication, weakness, pain, or other vague discomfort in the face or in one extremity, or merely in the distal part of an extremity. The face at the same time may show transient weakness, the leg may be dragged a trifle, and a little clumsiness may be noticed in the finer finger-movements. Speech very often is disturbed, presenting various slight aphasic indications. Any or all of these symptoms are likely to be most marked toward night or after fatigue. They indicate a local anemia of their respective cerebral mechanisms and a lowered nutrition that translates itself in paresthesias and paresis and foreshadows paralysis. In many cases the cortical irritation shows itself in twitchings in the face or in the extremities, or in a sudden thickness of speech.

Groups of such symptoms may appear several times, lasting at intervals for a day or two and then recede for weeks or months. Finally they return with more bruskness and severity, convulsions may ensue, the thrombosis is complete, and paralysis is permanently established.

This progressive loss of cortical circulation is to some extent a part of the physiology of senile involution. The blood-current becomes gradually weaker and the arterial channels progressively diminish in capacity. The heart loses its force; the fine, delicate, cortical vessels are most affected; circulatory stagnation in the brain-mantle is favored, nutrition fails, psychical and motor and sensory functions are reduced in activity, and dementia is inevitable. In the brains of the aged, multiple foci of softening are commonly encountered.

The paralytic state may thus be established suddenly with or without an apoplectic state, or may advance by steps and be preceded by numerous warnings and significant symptoms. Softening once established has the tendency, as has already been indicated, to invade adjoining cerebral areas, related through the arterial supply. Hemiplegia and the hemiplegic state are common results. In cases of sudden onset, especially if marked by an apoplectic seizure, the hemiplegia is usually complete and severe. In cases of less active onset, and especially in those of gradual development, the functional loss is less profound and there is a greater tendency to subsequent recession, leaving only a monoplegia or a monoparesis. The paralysis may be monoplegic from the first, and softening is the most common source of the cerebral monoplegias. In the same way aphasia alone may indicate the cortical disease. Persistent aphasia in all its varieties, nine times out of ten, is due to cerebral softening. In the same way the cortical areas of half-vision may be destroyed, or any given cortical function may be singly selected for abolition.

The paralytic state due to softening presents practically the same course and final deformities that mark it when resulting from cerebral hemorrhage (see p. 209). At first flaccid, the permanently paralyzed limbs show increased reflexes after about three weeks, and the usual contractures are developed. If the paralysis in a given member remain unimproved at the end of the first week, it is likely to be permanent. The tendency to recession, to progressive improvement, is by no means so marked as in hemorrhage. While the traumatic shock to the brain is less, the destruction is relatively greater.

Sensory disturbances, which in hemorrhage ordinarily disappear promptly, are commonly persistent in the paralytic state after softening. Their persistence indicates a cortical lesion unless hemiplegia and hemianesthesia coincide, when the sensory crossway is involved, whatever be the lesion, hysteria being excluded. The sensory disturbances, after cortical softening, are paresthetic, not anesthetic. This is due to the stratification of cortical function, sensation, muscular sense, and motion being represented at increasing depths in the sensorimotor zone, as described on page 162. That sensation is not abolished, but is only disturbed, is due to its probably complete bilateral representation in the hemispheres.

Course.—As cerebral softening is a brain accident, the result of arterial disease, like hemorrhage, it may present all gradations of severity and extent and numerous clinical forms. Cases marked by abrupt onset with a well-marked apoplectic phase may sink into coma and die. In proportion as the onset is gradual the tendency to immediate death is lessened, excepting in those instances where progressive invasion of vascular territories shows that the thrombotic process is advancing toward the circle of Willis. The paralysis or aphasia once definitely developed, there is still a tendency to improvement, which is less marked than in corresponding cases of cerebral hemorrhage. Should the softened focus become infected, an acute purulent encephalitis is likely to carry off the patient. This complication is marked by elevation of temperature, sometimes by somnolence, usually by convulsions referable to irritation in or about the diseased area, and is frequently associated with a pneumonia or an acute bedsore. Months and years after the onset of the disease the softened focus still constitutes an irritant brain-lesion which may cause epileptoid convulsions. It may have a bad influence on the integrity of the mind and be followed by insanity. The hemiplegic state, when once developed, presents the common features and indications described in the preceding chapter. When multiple softenings occur, the clinical picture is much modified. A right facial paresis, with aphasic symptoms, may accompany a left brachial monoplegia.

Diagnosis.—The diagnosis of cerebral softening often presents many difficulties and sometimes is impossible. In the great majority of cases, however, a careful scrutiny of all the data enables us to make a positive diagnosis, and in a fair share of the remaining cases a strong presumption can be established. The question primarily concerns the arterial occlusion. After the age of ten and up to forty a paralytic attack suggests embolism or syphilis. Endocardial disease, a recent history of acute rheumatism or infectious fever, speaks strongly for embolism. Practically a diagnosis of embolism can not be made in the absence of cardiac symptoms. After the age of seventy years the presumption is again in favor of softening, but from forty to sixty-five or seventy hemorrhage is the commoner cause of paralysis. A history of syphilis, plumbism, or alcoholism suggests softening.

The mode of onset may clearly indicate softening. Prodromata and the gradual paralytic invasion, localized spasms, monoplegias, aphasias, and limited paresthetic areas are indicative of softening. The disproportion between the paralysis and the apoplectic features suggests softening. A complete hemiplegia of rapid development without a stroke can hardly be due to hemorrhage. A pale face, weak heart, normal temperature, and practically undisturbed consciousness all favor softening as the cause of a paralytic attack. Multiple palsies and bilateral palsies, especially if symmetrical, are usually due to softening. Softening occurs often during sleep and under conditions of low arterial tension, except in embolism. After the paralysis has been established it is likely to persist, though in children and young adults it may notably improve.

Many of the clinical features of softening are produced by tumors, but ordinarily new growths have a more insidious course and present the cardinal symptoms of headache, vomiting, optic neuritis, and vertigo. Cerebral abscesses usually furnish a history or evidence of injury or otitis, but a softened area may be infected and break down into an abscess. It is with cerebral hemorrhage that softening is most confused. They have many common points. For instance, syphilis and alcoholism may cause both, and both have apoplectic onsets and paralytic sequels. In some instances the differential diagnosis can only be made out in the light of the subsequent course of the disease, and in a small number of cases it seems quite impossible to make it. The following table of probabilities will serve to show the direction a presumptive diagnosis should take:

TABLE OF DIFFERENTIAL PROBABILITIES IN CEREBRAL HEMORRHAGE AND THROMBOSIS.

	HEMORRHAGE.	THROMBOSIS.
PREDISPOSING CONDITIONS.	Frequent before three years of age, and between forty and sixty.	Common in old age and in young adults.
	Peri-arteritis and miliary aneurysm the usual antecedent.	Endarteritis, atheroma, endocarditis, and cachexiæ.
	Heredity often marked.	Heredity rare.

TABLE OF DIFFERENTIAL PROBABILITIES IN CEREBRAL HEMORRHAGE AND THROMBOSIS.—(*Continued.*)

	HEMORRHAGE.	THROMBOSIS.
INCITING CONDITIONS.	High arterial tension.	Low arterial tension.
	Excitement, effort, or shock.	Rarely excitement or effort, except in embolism. Sleep favors it.
ONSET CONDITIONS.	No prodromata.	Prodromata common.
	Sudden stroke usual.	Complete stroke rare.
	Coma marked.	Coma slight or wanting.
	Rectal temperature reduced, and surface temperature elevated on the paralyzed side.	Temperature usually unchanged.
	Congested face ; respiratory difficulties.	Pale face : no respiratory disturbance.
	Pulse slow, full, bounding.	Pulse weak, soft, often rapid.
	Motor loss usually hemiplegic and fully developed at once.	Motor loss often monoplegic and inclined to extend.
	General convulsions common.	Limited convulsions common.
COURSE.	Rapid improvement in motion.	Slow motor improvement. Extension of paralysis often observed.
	Foot usually gains more rapidly than hand.	Foot often gains less than hand.
	Anesthesia usually fleeting.	Paresthesia persists.
	Persistent aphasia exceptional.	Persistent aphasia and other cortical symptoms common.
	Postplegic athetosis, trembling, and chorea common.	Postplegic athetosis, trembling, and chorea uncommon.
	Postplegic convulsions rare.	Postplegic convulsions common.
	Spasmodic weeping and laughter common.	Spasmodic weeping and laughter exceptional.

Prognosis.—Cerebral softening is an accident following such a wide variety of diseases and conditions which provoke the endarterial process of thrombosis that prognosis can not be generalized. Every case has its own indications. The tendency to immediate death is less than in hemorrhage, but the appearance of pneumonia, or an acute bedsore, or a sudden elevation of temperature, even of moderate degree, indicates a grave complication and a probable fatality. In embolic cases, if it is probable that the embolus is infected, as in infectious endocarditis, diphtheria, and the exanthemata, the outlook is much darkened by the probability of acute infectious encephalitis being set up in the softened area, to be followed by abscess and probably by death. Advanced years are against the patient. In every case the prognosis should be held in reservation for a week until it is evident that the thrombosis is not spreading and that local infection has not occurred. The temperature is here a valuable guide. Persistent severe convulsions commencing early, perhaps present at the onset, are of grave significance. They point to involvement of the cortex and meninges on the one hand, or of the lateral ventricles on the other. The secondary implication of the meninges or ependyma over the softened area is usually limited, but in infected cases it may lead to a generalized inflammatory process of the utmost gravity. When the first fortnight has passed, the paralytic state may be considered established. Contractures and deformities are thereafter developed, as in hemorrhage. The hemiplegic state presents nothing dissimilar to that following arterial rupture, and has been described in a previous chapter. The condition presented by a given case of softening at the end of the first month is likely to be permanent. This is especially true after middle life. There is also the possibility of epileptoid attacks following at any time, and the persistence of the endarterial disease or its generalized presence constitutes a continual menace. This is particularly true in multiple softenings and in the bilateral forms such as that which furnishes a pseudobulbar palsy.

Treatment.—The treatment of cerebral softening to be efficient must antedate the occurrence of thrombosis. In a word, it must be prophylactic. In another word, it must be the treatment of the arterial disease. When the arterial current is cut off we yet have to deal with the basic disease in order to prevent an increase of the thrombus or its repetition and to cause, if possible, its diminution. When called at the onset of the softening in the early hours of the attack, if hemorrhage can be excluded, the treatment consists of maintaining a masterly inactivity. The patient's position should be horizontal, to favor the cerebral circulation; the flagging heart may be encouraged with strychnin and relieved with the nitrites given frequently, which also favor increased circulation in the exsanguinated brain territory. Small quantities of nourishment should be administered and the functions of the bowels and bladder supervised. If hemorrhage can not be excluded, the same course is still advisable, but if hemorrhage is diagnosed, the opposite plan of treatment for that condition, already described, should be instituted. Purgation and venesection can not benefit a cerebral territory already exsanguinated. In cases of embolism, cardiac repose is to be encouraged that other particles may not emigrate. Bromid, to control the convulsions, may often be required. The further management of the case is that of good nursing. The arterial state must never escape attention. Its amenability to treatment governs the outlook for the patient and the probability of recurrence. When the paralytic state is established, its management is the same as that laid down in the previous chapter, and for the terminal monoplegia or hemiplegia the indications are likewise identical. The treatment of aphasias and the development of the opposite-sided speech-centers have been described in the chapter on Aphasia.

CHAPTER VIII.

DISEASES OF THE CEREBRAL VEINS AND SINUSES.

Anatomical Considerations.—The blood entering the cranium by the internal carotids and vertebrals after irrigating the encephalon makes its exit mainly by the internal jugular veins. The return circulation from the ventricular portion of the cerebrum and the callosal portion of the hemispheres is by the Gallenic veins and inferior longitudinal sinus, all of which empty into the straight sinus. From the convexity the pial veins run upward and open into the superior longitudinal sinus in a forward direction against the slow blood-current of this dural channel. Here the circulatory conditions are rendered still more unfavorable by hydrostatic pressure, by the presence of trabeculæ in the sinuses which impede the flow of blood, and by venous retardation during inspiration. The cerebellar veins empty mainly into the lateral sinuses. Into the dural sinuses also empty many veins from the face and scalp. The facial vein communicates with the cavernous sinus through the ophthalmic vein. The veins of the nasal vault open into the anterior extremity of the superior longitudinal sinus. Numerous veins of the scalp along the median line have a similar outlet. Veins from the mastoid process and its cutaneous surface enter the lateral and

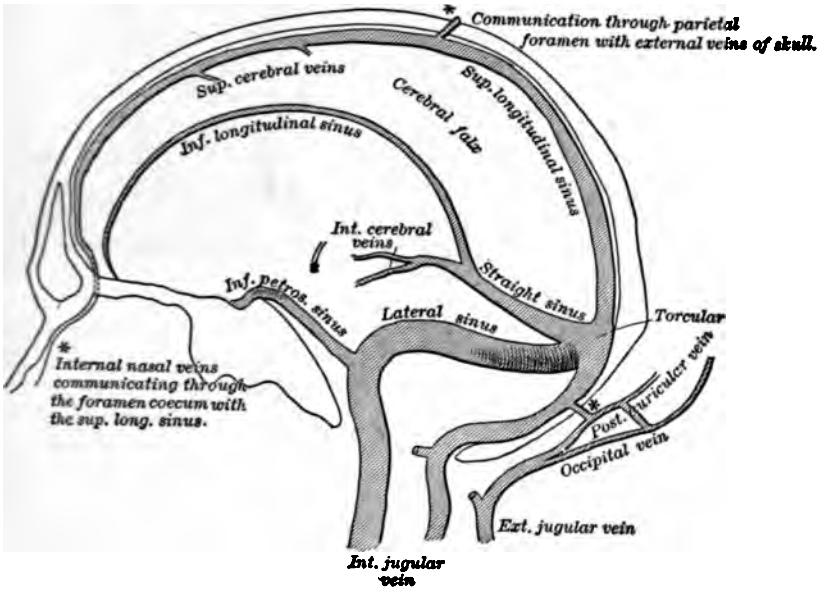

Fig. 88.—Diagram showing the communications existing between the superior longitudinal and lateral sinuses and the external veins, indicated in the figure by * (Leube).

petrosal sinuses, and the occipital and posterior auricular veins are connected with the lateral sinus. In addition, many veins of the cranial diploë discharge into the sinuses. Finally, the sinuses connect with the veins of the spinal canal.

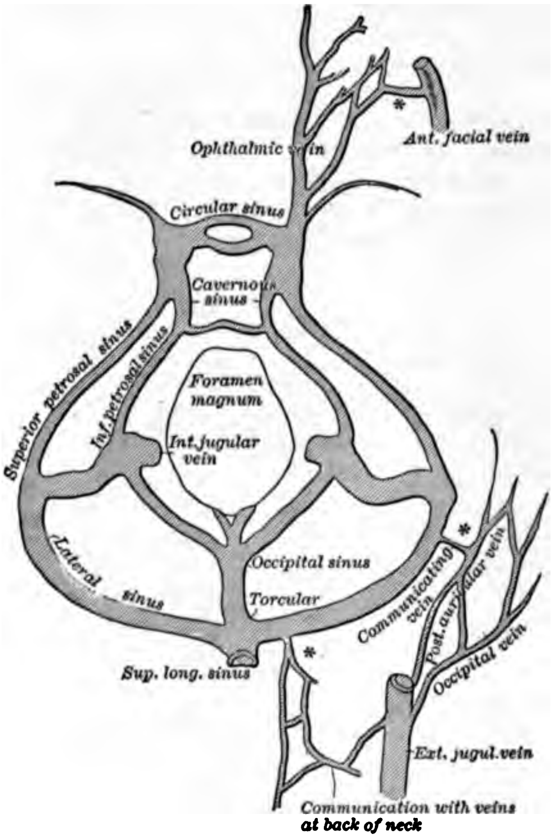

Fig. 89.—**Diagram** showing the communications existing between the lateral and cavernous sinuses and the external veins, indicated in the figure by * (Leube).

The cerebral veins are subject to the same lesions that influence veins elsewhere, but we are only called upon to consider phlebitis and venous thrombosis. They are usually associated and, practically speaking, severe cerebral symptoms are alone produced by thrombi. Thrombosis may occur in the pial vessels of the convexity and extend into the longitudinal sinus, or, beginning in the sinus, may invade the cortex and give rise to localized softening and focal manifestations. The entire subject may be conveniently described under the head of Sinus Thrombosis.

SINUS THROMBOSIS.

Intracranial sinus thrombosis occurs more frequently even than thrombosis in the pelvic veins, or in those of the lower extremities. It is favored by the sluggishness of the venous current and the other anatomical peculiarities above indicated. Septic invasion is also extremely liable from the relation of these channels to the cavities of the nose, throat, and ear, and to the frequently traumatized surface of the face, neck, and scalp. It occurs in two forms,—the marantic and the infective.

Marantic sinus thrombosis, or *primary thrombosis*, is a local condition occurring usually in the superior longitudinal, rarely in the lateral, and very rarely in the cavernous sinus. It occurs in debilitated states, and is most common at the two extremes of life. Exhausting diseases, weakness of the heart, and in general any cachectic state predispose to it. Prolonged illness, as from diarrhea, typhoid fever, pneumonia, phthisis, cancer, malaria, the anemias, etc., often precede it. Under these conditions there is a tendency to fibrin deposit and thrombus formation which, once started, is likely to extend. Should it commence in a cortical vein, or extend from a sinus to the brain-surface, Jacksonian fits may be produced.

Usually on post-mortem examination the thrombus is found to involve several sinuses and their tributary veins. When the entire lumen of a cortical vein is blocked, the drainage of its territory is prevented and localized edema, punctate hemorrhage, and red softening follow. In the same way edema occurs in the superficial parts of the face and head which drain into an occluded sinus. Swelling about the eye and exophthalmos, with retinal thrombosis and epistaxis; swelling about the mastoid, over the vertex or occiput, is produced by the thrombotic closing of the sinuses respectively related to these regions. When the lateral sinus is involved, the thrombus may extend down the internal jugular and be found as a firm, palpable cord in the neck. In marantic thrombosis the clots are firm and non-adherent to the walls of the vein or sinus,—that is, they are not inflammatory. They tend to organize or resorb and do not disintegrate. Reëstablishment of the circulation in the sinus is therefore possible, and usually takes place in long-standing cases, but in cortical veins, if cerebral softening occurs, there is no tendency to circulatory restoration.

The **symptoms** of marantic thrombosis are those of venous stasis, localized edema, and disturbed brain-function following upon exhausting diseases. As the longitudinal sinus is usually affected there is often epistaxis and disturbance in the leg-centers, indicated by weakness, tremors, paralyses, and spasms in the lower extremities. Convulsions in children are commonly encountered, and may be limited or monoplegic when a cortical vein is invaded. Meningeal irritation often shows itself in rigidities, retracted neck, and vasomotor disturbance.

The **diagnosis** of marantic thrombosis is usually obscured by the overshadowing picture of the preceding illness, and in those cases where external edema does not point the way it is rarely deciphered during life. There can be little doubt that it furnishes some of the cases of cerebral palsy in childhood. Convulsions coming on late in the history of exhausting illness, especially in children, should direct attention to the possibility of sinus occlusion. If meningeal or focal symptoms are present, coupled with local edema about the eye or face, over the vertex, in the mastoid or occipital region, a diagnosis may be made.

The prognosis will depend upon the nature of the initial illness, the probability of cortical softening, and the vital prospects. When the cachexia or exhausting disease can be controlled and life maintained, the tendency to resorption of the clot presents a favorable outlook, except for the softened areas of brain-tissue. When these have been produced, permanent disability of a motor or mental character, or both, may be expected. The *treatment* is that of the general condition.

Infective sinus thrombosis, *inflammatory thrombosis or secondary thrombosis*, is the result of the invasion of pathogenic infectious organisms. It is, therefore, a secondary process and occurs usually in adults. Generally it is located in one of the paired sinuses and in the one nearest the infection atrium. It is likely to produce meningitis or give rise to cerebral abscess, or both, and it is frequently attended or followed by systemic infection and pyemia. It arises from septic traumatic conditions of the face, scalp, cranial and facial bones, and from septic processes in the nose, mouth, pharynx, in the various bony sinuses, and in the middle ear and mastoid process. By far the most common source of infection is suppurative middle-ear disease. Anthrax of the face and lips, facial and scalp erysipelas, dental caries, and carbuncles have been noted as causes. The sinus always becomes infected by extension of the septic process to it, either by direct invasion or by propagation along a venous tributary. Inflammation of the sinus-walls is followed by thrombotic coagulation of the contained blood, and a septic plug is formed that closely adheres to the diseased and softened vessel.

The septic, soft, and disintegrating thrombotic clot, swarming with pathogenic and pyogenic bacteria, sets up infection of the immediate territory, and, yielding particles to the blood-stream, often induces disseminated and systemic infection. Thus arise the numerous pyogenic foci throughout the body that are frequently present. The thrombus once formed is prone to extend, and the internal jugular is often invaded, presenting itself in its upper third as an indurated cord that can be palpated in the neck. Even the superior vena cava has been invaded. The sinus-walls being softened, the neighboring leptomeninges are infected and a localized or disseminated septic meningitis is added. In a similar way the infective process travels along the veins into the substance of the encephalon and sets up abscesses. Macewen thus tabulates the principal differences between marantic and infective sinus thrombosis:

SINUS THROMBOSIS.

MARANTIC.	INFECTIVE.
1. Chiefly affects the longitudinal sinuses.	1. Chiefly affects the paired sinuses.
2. The clots tend to organization or are absorbed.	2. Clots tend to purulent disintegration.
3. Hemorrhages into cerebral cortex in about half the cases.	3. Hemorrhages into brain or cerebellum seldom occur.

4. Tendency to produce brain softening.	4. No tendency to brain softening.
5. There is seldom purulent infection as a sequence.	5. Purulent infection common from septic or infective emboli.
6. No accompanying leptomeningitis, cerebral or cerebellar abscess.	6. Often coincident purulent leptomeningitis, cerebral or cerebellar abscess.

The symptoms of infective sinus thrombosis are *local* and *systemic.* The local ones are due to circulatory disturbance, such as circumscribed edema and brain symptoms. They will be given in detail in the description of thrombosis of special sinuses. The systemic symptoms are those of septicemia; intense headache, often localized at the seat of disease; vomiting, fluctuating and remittent temperature; small, thready pulse; rigors, profuse perspiration, dry tongue, anorexia and diarrhea, or constipation.

Depending upon the preponderance of symptoms and their grouping, the septicemia shows different clinical varieties. When the lungs are first or mainly involved by the plugging of pulmonary vessels, localized or diffuse pain is occasioned, cough is induced, and the expectoration may change to "prune-juice" appearance, and then becomes purulent, fetid, and extremely offensive as the pulmonary process increases. Abscesses form and gangrene occurs. In this way septic pneumonia also is induced.

When the brunt of the attack falls upon the abdominal organs, the typhoid type of septicemia, which closely mimics enteric fever and is sometimes mistaken for it, is presented. In another and much smaller group of cases meningeal symptoms dominate the picture and are actually due, in large part at least, to the infective leptomeningitis. All three of the symptom groups, or any combination of their various features, may be presented by the same case.

Infective cavernous sinus thrombosis arises from septic invasion, reaching the sinus usually by way of the ophthalmic vein. It may also be due to a forward extension of a septic process in the lateral or petrosal sinuses. The secondary meningitis to which it gives rise is basilar. Fractures of the cranial base and blows on the head have furnished its starting-point. Erysipelas of the face, especially about the eye and nose; abscess of the orbit; infections of the nasal, buccal, and pharyngeal cavities or of their sinuses; ulceration of the tonsillar glands, and caries or periosteitis of the facial, especially of the maxillary bones, have led to it.

Symptoms: One sinus is usually first involved, and the local manifestations are one-sided. There is, however, a pronounced tendency for the process to invade the opposite sinus, and then the case presents bilateral signs. Such a sequence is highly diagnostic. The first affected side may even show improvement through the establishment of the collateral return circulation before the second side is invaded. There is usually considerable pain of a supra- or infra-orbital neuralgic sort, and diffuse headache. Mental symptoms are wanting, unless meningitis is set up, and then delirium, hebetude, and coma may appear. When the sinus is plugged the return circulation through the ophthalmic veins is cut off. The orbital contents become edematous, the ocular globe is thrust forward, the lids are swollen, and the swelling extends to the nose, brow, and cheek. There may also be swelling on the same side of the pharynx. The optic disc is congested or choked, the retinal veins are distended, and pressure is exerted on the ocular nerves that enter the orbital apex. This causes more or less ophthalmoplegia. The third, fourth, sixth, the ophthalmic division of the fifth, and the optic nerves are more or less affected. Ptosis, strabismus, pupillary stasis, and defective vision in varying degrees are thereby added to the exophthalmos.

The invasion may be abrupt or insidious and the disease may last from a few days to several months, but infective cases are practically fatal. When the second eye is involved, it usually is very rapidly affected. The appearance of basilar meningitis and the development of septicemia add immediate gravity to the already serious condition.

Infective lateral sinus thrombosis is the form most frequently encountered. Its origin is nearly always in a septic condition of the middle ear. The petrosal sinuses and the internal jugular are usually invaded. It is commonly encountered in young adults, and is rare in the two extremes of age. While ordinarily due to middle-ear disease, it may arise from a mouth or throat infection by way of the Eustachian tube and tympanum, from extension of thrombosis in other sinuses, from basilar fractures involving the petron, and from infections about the occiput, nucha, and mastoid.

Symptoms: The lateral sinus is usually affected from a chronic middle-ear suppuration which has caused more or less erosion of the tympanic bony structure. An acute process may cause it, but this is exceptional. In the chronic cases of purulent otitis media it is a constant menace. Frequently there is a lessening or cessation of the ear-discharge, pain develops in the ear, and headache follows. The fluctuating temperature mounts up, and vomiting and rigors indicate the involvement of the sinus or an intracranial extension. Local *signs* of lateral sinus thrombosis depend on the obstruction of its lumen and the location or extent of the thrombus and phlebitis. Occlusion of the sinus, blocking the inlet of the mastoid vein, gives rise to a circumscribed edema extending from the auricle over the mastoid. Pain on percussion of the mastoid is present only when the bone or periosteum is inflamed. If the thrombosis is situated lower in the sinus and shuts off the condylar veins, through which the superficial circulation of the lateral lower occipital region drains, a brawny hardness and edema may be made out in the upper part of the posterior cervical triangle. Should the phlebitis extend into the internal jugular, extreme local tenderness is found over the upper portion of this vessel on palpation, or may be experienced by the patient in swallowing. The head is usually inclined to the affected side to lessen muscular pressure on the jugular. When the thrombosis follows down into the jugular, it may be easily palpated as a firm, cord-like structure. Disintegration of the thrombus may cause it to disappear in a few days. The lymphatic glands in the neck are frequently engorged and easily palpable. Gerhardt has pointed out that during inspiration the external jugular vein on the affected side is less prominent. This is due to the occlusion of the internal jugular, which allows rapid drainage of the external branch into the common trunk. If, however, the thrombus extends into the common trunk, the external vessel is then engorged and more prominent than on the sound side.

If the inflammatory thickening at the jugular foramen is sufficiently great, it involves, by extension or pressure, the cranial-nerve trunks, which make their exit at this opening. Pneumogastric, spinal accessory, and glossopharyngeal symptoms are then produced. Respiratory, laryngeal, cardiac, and vocal disturbances; difficulty in swallowing, spasm or paresis in the sternomastoid and trapezius point to this condition. Abscesses sometimes form in the neck, under the sternomastoid or in the nuchal region.

Almost from the first there are distinct cerebral symptoms. The cephalalgia is attended by somnolence, which may deepen into coma. Delirium is often an early symptom. Phlebitis is likely to extend into the temporosphenoidal lobe and cerebellum, giving rise to diffuse inflammation or rapidly producing softening and abscesses. At the seat of the sinus thrombosis the softened dural wall no longer protects the soft meninges, and a localized septic meningitis, with a tendency to become diffuse is occasioned. Even the bone under the sinus is eroded, and definite discolorations are left, both on the cranial wall and on the cerebral surface, to mark the site of the sinus disease.

A disease presenting so many complications and possibilities necessarily lacks clinical uniformity. While lateral sinus thrombosis is usually confined to one side, it may propagate itself into the venous channels of the opposite side and infect both internal jugulars. The appearance of cerebritis, meningitis, or intracranial abscess greatly reduces the life chances. Septicemia is particularly likely to develop, with its own serious import. The virulence of the infection, however, seems to vary between wide extremes. Some cases run a rapid course to fatal termination in four to seven days, others last weeks and months, and exceptional ones may recover. The gravity of the disease can not well be overestimated.

Infective thrombosis of the longitudinal sinuses is extremely rare, while marantic thrombosis finds its most common location in these venous passages and especially in the superior one. Infectious conditions in the nasal vault, in the scalp, and in the cranial diploë of the vertical region are capable of extension to the superior longitudinal sinus. Phlebitis may then extend to the cortical veins. Occlusion of the sinus leads to local edema in the scalp and probably in the brain also, but the collateral circulation obviates any serious results from this mechanical feature. The danger lies in the likelihood of septic cerebral phlebitis and septic meningitis. Usually the obtrusive meningitic features predominate, and suggestions of cortical phlebitis and even of abscess formation are obscured or overlooked.

The **treatment** of infective cranial sinus thrombosis is primarily surgical. The infection atrium is to be determined and rendered thoroughly aseptic. Whenever possible, it must be eradicated. In the case of the cavernous sinuses little more can be done by the surgeon, but Dwight and Germain[1] have reported one instance in which the cavernous sinus was reached surgically with immediate improvement in the patient's condition, though with fatal termination ultimately. A previous case by Hartly was entirely successful. Aggressive interference is allowable, and indicated when the lateral and longitudinal sinuses are invaded. Many cases of lateral sinus thromboses have probably been saved by prompt and radical interference. The sinus has been opened, the infective thrombus removed, and abscesses in the temporal lobe and in the cerebellum drained. Often as a preliminary measure the mastoid antrum and the middle ear have been surgically dealt with, but when there is evidence of sinus thrombosis it is a waste of time and opportunity to stop at this step.

Local applications of heat and cold to the head may be used, and serve sometimes to relieve the headache and modify the delirium. In view of the septic nature of the disease, supportive measures are emphatically indicated from the first. Against the septicemia we may bring measures to bear that favor elimination by the skin, bowels, and kidneys. The administration of antiseptics, such as the salicyl preparations and the mercurials, are of doubtful value, but are strongly advised by many and should not be omitted.

CHAPTER IX.

CEREBRITIS, ENCEPHALITIS, AND ABSCESS OF THE BRAIN.

INFLAMMATION of the brain proper may be *local or generalized, acute or chronic.*

Acute Localized Cerebritis.—**Etiology.**—The brain is subject to inflammatory processes the same as any other parenchymatous organ, yet *generalized cerebritis* is rare, and only recently have we learned to recognize it. *Localized acute cerebritis*, on the other hand, occurs with frequency, but is practically, if not invariably, a secondary condition. *Meningitis* always entails some underlying cerebritis. A circumscribed cerebritis occurs about a *hemorrhagic focus* or spot of *thrombotic softening* if infection is added. Without the additional infection such as is furnished by septic endocarditis or other infective focus the zone surrounding a cerebral tumor, hemorrhage, or area of softening is one of congestion, edema, and pressure necrosis. *Blows on the head* may cause localized meningitis and cerebritis at the point of violence, or on the opposite side of the head by the action of contre coup. In some cases the local cerebritis alone follows, and this may only affect the white matter below the cortex. Apparently the traumatism favors the local action of bacteria by reducing the resistance of the tissues, as proved by Ehrnrooth in an interesting series of experiments.[1] Cranial *fractures*, *punctured wounds*, and perhaps severe *concussion* may cause it. *Bone disease*, *septic processes* in the diploë, and in the cranial bony and venous sinuses, may lead to cerebral inflammation and be followed by abscess. Infection of the brain after *operations* sometimes causes a diffuse cerebritis that may involve an entire hemisphere. *Hernia cerebri* is a manifestation of a septic inflammatory process. *Sunstroke*, heat-stroke, and alcoholism seem capable of inducing cerebritis, or at least so modifying the cerebral nutrition that dementia may follow and sclerotic brain-tissue be found postmortem. To Van Giesen we owe a knowledge of the cellular changes following insolation and their probable relation to an acute auto-intoxication.

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[1] "Boston Med. and Surg. Jour.," 1902.

In certain conditions the inflammation falls on definite cerebral structures. In this way arise the clinical symptom groups described under the terms *acute polio-encephalitis superior* and *acute polio-encephalitis inferior*, when the cranial nuclei are invaded in the upper or lower groups, furnishing acute nuclear ophthalmoplegia in one instance and acute bulbar palsy in the other. These have already been described with the diseases of the cranial nerves. Strümpell alleged an acute inflammatory process in the cellular cortex, especially of the motor regions, analogous to that of poliomyelitis as the explanation of many cases of cerebral palsy in children. To this he applied the term polio-encephalitis, a name given also to the inflammations of cranial-nerve nuclei, making an additional adjective necessary. It is, therefore, sometimes called polio-encephalitis anterior or corticalis. There is little pathological evidence of this form of cerebritis, and the clinical manifestations of the attack are consistent with the idea of a vascular lesion. As there is a growing belief in the vascular origin of poliomyelitis, the analogy, if not the identity, of the process is still maintained, and probably it is associated with an acute arteritis of an infectious sort in both instances. Reymond[2] reports what appears to be a typical case in a child of seventeen months. The microscopical changes were certainly of inflammatory origin and the disease was not limited to the motor fields.

The pathological anatomy of the various conditions which have been assembled under the head of acute localized cerebritis shows at first a high degree of vascular engorgement, edema, and punctate hemorrhage. The inflamed tissue presents above the level of the surrounding part, and is often slightly softened. Its reddish color and yielding consistency have led to its description as a red softening. It has much the macroscopical appearance of embolic red softening, with which it was long confused. Leukocytal infiltration of the brain and the disintegration of neuroglia, myelin, and nerve-cells may induce a pultaceous and even creamy consistency. A nidus ripe for infection is formed, and when this occurs abscess formation is prompt. Later on, if not infected, there is usually more or less shrinking of the inflamed mass, absorption of the necrosed elements, proliferation of the connective tissue, and sclerotic thickening which may reach a leathery toughness. Small foci may in this way become cicatrized. Finally, after years, it may be impossible to say whether inflammation, hemorrhage, or thrombosis was the initial feature of the cerebral defect.

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[1] "Rev. Neurolog.," Aug., 1900.

[2] "Jahrb. f. Kinderheilk.," Bd. xliv.

Symptoms.—The indications of acute localized cerebritis are obscure. Often associated conditions, such as meningitis, overshadow it in the clinic. Whatever be the seat of the cerebral inflammation, there is usually headache, sometimes vomiting, and rarely optic neuritis. Delirium and somnolence may be present. Involvement of special sensory or motor convolutions or pathways gives rise to localizing features marked by defect or disturbance of function. The clinical picture, therefore, always strongly suggests meningitis, and the *treatment* and *prognosis* are practically the same as in that condition.

Acute Hemorrhagic Encephalitis.—Strümpell, in 1889, and shortly afterward Leichtenstein, in 1890, called attention to cases of diffuse hemorrhagic encephalitis. In 1895 Oppenheim [1] reviewed the literature of the subject, pointed out the intimate relation of the disease to Wernicke's polio-encephalitis superior and to Strümpell's polio-encephalitis anterior, and added six cases, several of which recovered. Other cases have been reported by Fürbringer, Putnam, Freyhan, Neuwerk, and Brie, all of which are quoted by Putnam.[2] Brie [3] has since reported a second instance, with full autopsy and bacteriological investigation. There can be little doubt that numerous cases have been mistaken for meningitis. Anatomically the disease is marked by multiple, non-suppurative, inflammatory foci showing congestion and punctate or massive hemorrhages, leukocytal infiltration, and localized destruction of brain-tissue.

Etiology.—Regarding the causation of acute hemorrhagic encephalitis there is much to be discovered. Most of the reported cases followed influenza. Putnam's last case followed the mumps, and erysipelas, diphtheria, typhoid, typhus, and malaria [4] have been followed by localized hemorrhagic encephalitis. The writer has seen two cases after influenza, one after pneumonia, and one with some unknown infection associated with acute nephritis. It is a matter of speculation whether various infections act locally or by their elaborated toxins. An interval, a sort of incubation period, is often noted between the infectious disease and the manifestation of the cerebral symptoms. Children and young adults furnish most cases.

Morbid Anatomy.—The meninges commonly are normal in appearance. There is usually some increase of cerebral fluid, and the ventricles may be largely distended by blood-tinged contents. The choroid plexuses and the vessels generally are engorged. Scattered throughout the brain, but principally in the white matter of the hemispheres and in or about the basal ganglia, are foci of hemorrhagic, softened, infiltrated brain-tissue. In some instances the hemorrhage is massive, in others there is but a slight hemic coloring. Microscopically the blood-vessels are found distended, the lymphatics filled with blood-cells and leukocytal elements, and at various points they are ruptured into the surrounding brain-substance.

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[1] "Deut. Zeit. f. Nervenheilk.," Band vi.

[2] "Jour. Nerv. and Ment. Dis.," Jan., 1897.

[3] "Neurol. Centralbl.," No. 1, 1897.

[4] Dana, "Medical Record," July 7, 1900.

The influenza bacillus has been found in these foci,[1] and there are some who think the process may originate in bacterial embolism.

Symptoms.—The symptoms of acute primary hemorrhagic encephalitis are not definite, and commonly suggest meningitis. A preceding acute infection may be followed immediately or after several weeks by headache, hebetude, vomiting, convulsions, or localized palsies. Sluggish pupils and squints are rather common. Sleepiness that tends to coma is usual, and rigidity of the neck has been repeatedly observed. The pulse may be slow, the temperature elevated, normal, or subnormal. The palsies, which may be multiple, declare the localized processes and have a tendency to vary in intensity from day to day. Pulse and respiration become shallow and frequent, the stupor deepens, bedsore may form, and death terminate the case in a few days or in several weeks. A fair proportion of cases, as insisted upon by Oppenheim and others, recover, almost, if not completely, after a tardy and anxious convalescence.

It will be seen that the disease is clinically obscure and the *diagnosis* has usually been made post mortem. During life it is usually confounded with meningitis. This is not of great practical moment, as the *treatment* is the same. The *prognosis* is grave, but rather more favorable than the early descriptions indicated. Gradual onset, comparative mildness of symptoms, and long duration of disease have a favorable import.

Chronic cerebritis, and *chronic encephalitis* are terms loosely applied to late and usually secondary conditions that are mainly sclerotic and degenerative in nature. This sclerosis may exist in disseminated patches or in large, circumscribed areas; it may involve an entire hemisphere or be largely confined to the gray matter of both half-brains. The portion of the encephalon thus affected is disturbed in its function, which is usually greatly impaired or entirely abolished.

Abscess of the Brain.—Brain-abscess is always secondary, and is due to the invasion of pyogenic bacteria.

Etiology.—In a rough way we may say that all the causes of infective *sinus thrombosis* and acute localized *cerebritis* are competent to produce brain-abscess, as the first step in its development is inflammation. *Suppurative middle-ear disease* furnishes almost two-fifths of the cases, and *head injury* about one-fourth. About one-sixth of the cases are due to *general pyemic states.* Of this number purulent pulmonary diseases furnish the great majority. Septic inflammation in the nasal and pharyngeal spaces, brain-tumors, infected cerebral hemorrhages, and infarcts furnish a small contingent. In rare cases the oïdium albicans has been found in the abscess and traced through the blood-vessels into the nose. In others the only pathogenic bacterium present is the tubercle bacillus. Actinomyces have also been encountered. *Males* are about three times as subject to abscess of the brain as *females*, and five times as frequently affected with the traumatic variety. More than half of the cases occur between ten and thirty years of *age.* Koerner states that in Prussia about five per cent. of all deaths between the ages of ten and twenty are due to cerebral complications of otitis, mainly abscesses.

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[1] Putnam, *loc. cit.*

Pathological Anatomy.—The most common *seat* of brain-abscess is in the temporosphenoidal lobe, due, doubtless, to the relation of the venous circulation of this part of the brain and of the middle ear with the petrosal sinuses. In decreasing frequency follow the cerebellum, the centrum ovale, the pons, the occipital lobes, the parietal lobes, and the frontal lobes. The *invasion pathway* is often obscure, but the arterial route has been demonstrated in some instances, the perivascular spaces in others. The common sequential relation of middle-ear disease, sinus thrombosis, and brain-abscess calls attention at once to the venous channels as entry-way for the pyogenic bacteria. The extension of cerebral phlebitis from a sinus thrombosis was repeatedly mentioned in the preceding chapter as inducing cerebral softening and abscess formation. Frequently the abscess is *single* and of a size varying from a pea to a hen's egg or even to a larger size. In the regions of latent lesions, especially in the frontal and occipital lobes, an abscess may attain very large dimensions and contain many ounces of pus. Very commonly *multiple* brain-abscesses are encountered. In ear disease, abscesses, both above and below the cerebellar tentorium, are generally encountered, and failure to explore the cerebellum after the evacuation of an abscess in the temporal lobe has lost lives. When the infection arises from purulent lung disease or septic endocarditis, the left side of the brain is more affected for the same reason that embolism favors the left hemisphere. Under these circumstances, and in the case of infection from typhus, enteric fever, and other general pyemic states, numerous small abscesses may be found. In this way the brain may be fairly riddled with multiple abscesses. Relatively the *gray matter* of the brain is less liable to abscess formation than the white substance, and the cortex is often preserved over an extensive underlying abscess. Rarely a fistulous tract communicates with diseased bone, or reaches the outer surface of the cranium, or discharges into the nasal fossæ. On the other hand, no connection may be discernible between the infection and the abscess. The *pus* varies with the nature of the infection and the age and character of the abscess. In some recent cases it merely saturates the softened tissues; in others it is encysted; in a third variety an encysted old abscess is found floating in a secondary, surrounding, more recent abscess, the walls of which are made up of infiltrated, purulent, softened brain-tissue. In the old cases the pus is thick, yellowish or greenish, and in about a third of them extremely fetid and offensive. Sometimes it is reddish from the admixture of blood. The pus-corpuscles and leukocytal elements vary according to the age of the lesion. The streptococcus is usually found, but pneumococcus, staphylococcus, bacillus pyocyaneus, bacillus tuberculosis, and the bacteria of various mixed infections have been noted.

The majority of abscesses are *encysted*. When located near the surface, the meninges may form part of the containing wall. A beginning cyst-wall has been noted as early as the thirteenth day. Its thickness and consistency increase with age. It is made up by a proliferation of the neuroglial tissue and the fibrous structures of the brain. Usually delicate, in some instances it forms a glistening, resisting membrane of almost a horny consistency, and it may even calcify. The brain-tissue surrounding an abscess is usually more or less softened, and if infected rapidly breaks down, leaving the old abscess-cyst afloat. Rupture of an abscess by its steadily increasing contents, which is the usual ultimate accident, inundates the brain-tissue or breaks into the meninges, setting up a purulent process, or floods the ventricles and promptly terminates life.

Symptoms.—The symptoms of brain-abscess are usually indefinite, often very obscure, and sometimes entirely unnoticed by patient and physician. Different cases present the widest variety of clinical phases, depending upon the virulence of the infection, the rapidity of abscess growth, the location of the process, and the complications. Some cases run a rapid course to a fatal termination in a few days, and others present a latent period that may last months and even years, to end with rupture and death. We may, in some cases, make out *three stages*,—one of invasion, one of remission, and a terminal one of paralytic features. In other words, one of cerebritis, one of encapsulation and latency, and one of rupture, infection, meningitis, or ventricular inundation, and death.

The *invasion stage* presents the obscure picture of encephalitis. There is a low febrile movement marked by a vacillating temperature, which sometimes has a distinctly subnormal tendency, with a slow pulse. An intense and persistent headache lasting days, taken with the temperature, suggests meningitis. The headache is often circumscribed and may correspond to the location of the abscess, but as frequently is felt at a distant point. Rigors and profuse sweats indicate the septic character of the disease. A varying degree of leukocytosis is frequently found. Vomiting and constipation or diarrhea are often present. Papillitis or optic neuritis only rarely occurs. The mental state is one of torpor and indifference, or delirium may be present and the sharp "hydrocephalic" cry may be uttered. This stage lasts from two to ten days and may pass at once into the third stage or be followed by a remission.

The *remission stage* is gradually established by the subsidence of the disturbances that appear in the period of invasion. The headache, fever, vomiting, and mental irritation almost disappear or entirely cease. Occasionally the improvement is very prompt and complete. A *latent period* is thus produced that may extend almost indefinitely. The gradual increase in the size of the abscess and its encapsulation gives rise to very little disturbance. Its effect is that of a foreign body, and its presence is marked by much less disturbance than attends the growth of a solid tumor. At times there may be intense headache, vomiting, and even convulsions. An occasional temperature, or a tendency to a remitting temperature, sweats, and emaciation may show the hectic state and alone indicate the ever-present danger.

The *paralytic stage* is the usual termination of brain-abscesses that have presented a period of latency or remission. It is of sudden onset, and rapidly runs its course within a few days. It not infrequently promptly follows the invasion period, and the fatal mechanism is usually the same in both. In some cases this consists of a rupture of the abscess, causing a rapid infiltration of the cerebral capsule; an invasion of the medulla; a tearing through the cortex, setting up a purulent meningitis; or the inundation of the ventricles. There remain, however, a large number of cases in which these anatomical conditions are wanting to explain the sudden onset of this stage. Ordinarily an apoplectic stroke, with or without convulsions, takes place. Usually there are no premonitions or only the vague indications of cerebral mischief which have presented at intervals during the latent period. The apoplectic coma may be punctuated by Jacksonian fits, marked by conjugate deviation of head and eyes and some lateral weakness, and the patient may succumb in a few hours without regaining consciousness. Death, indeed, may be sudden. If the immediate effects of the stroke subside, a hemiplegia with marked rigidity, and often with spasmodic features, is progressively developed. The headache, the fever, and the symptoms of the early stage reappear or become intensified. Nystagmus, pupillary inequalities, and involvement of the ocular muscles appear and death is likely to follow, preceded by delirium and coma.

The *localizing* symptoms of brain-abscess are rarely prominent. Usually they are quite indefinite or entirely lacking. The location is often in the frontal, temporal, and occipital lobes, or in a cerebellar hemisphere, whence focal symptoms do not arise. As a rule, an abscess of the brain, due to disease of the cranial bones, lies subjacent to the original lesion. In the same way ear disease gives rise to abscess near the petron, but in rare instances the purulent collection has been found at a considerable distance, and even in the opposite half of the brain. Abscess due to embolic process from the heart or lungs is likely to be located in the capsular or Sylvian arteries, and gives rise to early hemiplegic features or disturbances of the motor cortex. The circumscribed headache sometimes furnishes a localizing feature, but it can not be relied upon. Macewen has noted a higher percussion note over the abscess than was yielded by the rest of the head, and Dana has confirmed this symptom in one case. Local tenderness and increased temperature may occasionally be made out. When the abscess is associated with septic sinus thrombosis, we have, in addition, the local superficial evidence of that disease. Even when the sensorimotor zone is invaded, the symptoms may be very slight. Abscesses may, however, yield as definite localizing features as any other encephalic lesion, and these then have the significance and value discussed under the head of Cerebral Localization. Involvement of cranial nerve-trunks, cerebellar symptoms, hemianopsia, Jacksonian fits, aphasias, and other localizing indications should always be carefully sought. Multiple abscesses also lead to symptoms of corresponding diversity.

Diagnosis.—The diagnosis of cerebral abscess depends very largely upon the history or presence of trauma or of an infective condition about the head and face or in the thorax. A chronic otorrhea, maxillary abscess, ozena, sinus suppuration, or purulent pulmonary disease, followed by headache, vomiting, delirium, stupor, slow pulse, vacillating temperature, and rigors, means encephalic invasion. These symptoms are common to meningitis and abscess, and the differential diagnosis can not always be made. Any evidence of a circumscribed process, however, favors the idea of abscess, and hence localizing symptoms become very important. The two conditions are often associated and a terminal suppurative meningitis is common in abscess. In latent periods the differentiation of cerebral abscess from a growth may be impossible; nor is it essential. The terminal stage, with its rapid onset and paralytic features, may readily be confounded with cerebral hemorrhage or softening if it occurs in middle life and is preceded by apparent health. When systemic sepsis is manifested, the purulent character of the encephalic process is less doubtful.

Abscesses resulting from ear disease, bone disease, and disease about and in the facial cavities—in other words, abscesses due to direct invasion—are ordinarily single. Abscesses resulting from pyemic states and from purulent thoracic conditions are usually multiple. Cerebellar abscesses are also usually multiple. The localizing diagnosis is made on the lines already laid down in chapters iii and iv of this part.

Prognosis and Treatment.—Suppurative disease within the cranium is always grave. Although a cerebral abscess may encapsulate and lie dormant for years, this can not reasonably be expected to take place, and even when it does, it constitutes a constant menace to life. Encapsulation does not necessarily check suppuration, and eventual rupture or secondary infection is the legitimate sequel. The late results are ordinarily fatal, and latent cerebral abscesses account for a certain proportion of sudden deaths.

The *treatment* of cerebral abscess should be prophylactic. Chronic suppurations in ear and nose should never be neglected and the utmost precautions should be taken, in the management of all wounds about the head and face, to prevent infection and sepsis. From these largely controllable sources the great majority of brain-abscesses and other endocranial suppurations arise. When the diagnosis is established, there should be immediate recourse to surgery. Of all encephalic diseases abscess promises the best results to surgical measures. Adequate drainage after opening the skull has been followed by brilliant success in numerous cases and in the hands of many operators. All other measures are inadequate.

CHAPTER X.

THE CEREBRAL PALSIES OF CHILDREN.

A NUMBER of varied developmental and acquired cerebral defects result in paralysis in childhood. They involve or destroy within the cranium some portion of the upper motor neuron, and give rise to clinical types that justify a separate description. Their importance and frequency have been recognized only within a few years, but it is now appreciated that in the early years of life cerebral palsies are fully as common as the spinal variety. Etiologically, they may be divided into three groups: (1) Those due to prenatal conditions; (2) those following birth accidents, and (3) those dependent upon disease or trauma after birth. Clinically, we also divide them into those showing unilateral defects, the hemiplegic cases, and those presenting bilateral defects, the diplegic cases.

Etiology.—One of the most important *prenatal conditions* resulting in infantile cerebral palsy is an actual deficiency of brain elements, a true agenesis. This may be marked by convolutional simplicity and a lessened number of cortical cells and pyramidal fibers. In other cases a part or the whole of a hemisphere, or of both hemispheres, may be lacking. Between these gross teratological defects and a condition in which the neuron units have diminished dynamical qualities or a lessened power of endurance, that in early life leads to their atrophic or sclerotic degeneration there is but a difference of degree. The lack of constitutional endurance, of capacity for growth, and of resistive power underlies many brain and cord diseases that present hereditary and familial characteristics. In other instances it furnishes a lowered resistance to toxic influences. In given cases unfinished children, lacking motor elements in the cortical mantles, and children born prematurely, before the pyramidal apparatus has been well established, present various degrees of motor and cerebral defect. The upper motor neuron is well formed only at the ninth intra-uterine month, and not completely developed until the second or third month after birth. Much defect in the upper neuron is always marked by spasticity and impaired motility in the muscles supplied by the lower or terminal motor neuron.

Rarely direct traumatism has affected the brain of the unborn child, which has presented hemiplegic contractures at birth. In other cases evidence of hemorrhage and softening has been found, and in many instances the probability of a prenatal meningo-encephalitis has been upheld by the presence of localized and diffuse sclerotic changes in the brains of new-born children. Inherited syphilis and other toxic conditions due to illness of the mother have been accused in some of these cases. Porencephalia is usually due to intra-uterine disease of the cerebral vessels.

The *conditions attending birth* frequently lead to brain-lesions in the child. The great majority of cerebral birth palsies occur in protracted labors, and consequently in primiparæ. A number of them follow precipitate labor and in both are due to violent compression of the fetal head. Comparatively few can be attributed to the use of forceps, and it is exactly in these cases that the labor is likely to have been tedious. Forceps accidents, however, can not be denied or overlooked, and the misuse of these powerful instruments is fraught with serious results to the skull and brain of the child. The facts, however, favor early skilful instrumental interference as compared with tedious labor.

The frequency of hemorrhage into the cerebral and spinal meninges during birth has been put on a sound foundation by the investigations of Litzmann, McNutt, and Spencer. It is found in the great majority of stillborn children and is the common cause of asphyxia of the newborn. Occasionally this blood comes from a ruptured longitudinal sinus, but most frequently from the pial vessels. Punctate and larger hemorrhages within the substance of the brain and cord are frequent. The results of these birth hemorrhages depend upon the quantity and location of the effused blood. Where death does not follow, all degrees of disability are encountered. Palsies follow the involvement of the motor cortex or its pyramidal tracts, but if the frontal lobes are seriously affected, idiocy is a consequence and is a frequent accompaniment of motor disturbance. Similar disturbance of the latent lesion territories of the brain may give rise to insignificant or very obscure symptoms. As a rule, the hemorrhage is basilar in location in vertex presentations and vertical in breech cases. The absorption and organization of these hemorrhages lead to a more or less diffuse sclerosis of the brain-substance that inhibits its growth and diminishes its functional capacity. In other cases in terminal stages large areas of softening and cyst-like formations are found that are indistinguishable from porencephalia. In about one-fourth of these cerebral birth hemorrhages the spinal cord is similarly affected. Some cases of syringomyelia may have this origin.

The *postnatal* causes of cerebral palsies in children, according to Osler's list, are hemorrhages, embolism, endo- and peri-arterial changes, encephalitis, and cerebral venous thrombosis. In other words, they are the same as in adults, and hemorrhage is more frequent than thrombosis. The relation of acute infectious diseases to embolism and arteritis only needs to be recalled. Traumatism and tumor formation cause some cases. Of encephalitis much has been said, and Strümpell claimed cortical polio-encephalitis to be the lesion in many of these cases, but the exact pathological process still escapes us owing to the paucity of early post-mortem examinations. It is just as likely that the lesion is primarily vascular. Gowers was the first to insist on the part played by venous thrombosis, and others, among them Osler, have added weight to the contention. This cerebral venous thrombosis is often a part of sinus thrombosis, to which it may hold a primary or secondary relation. It serves to produce softening, and sclerotic changes in the cortex and palsies follow. As many of these palsies start with convulsions, the question arises whether the convulsion is the cause or effect of the lesion. It may be either. There can be no doubt that the increased arterial tension of the convulsed state may cause rupture of cerebral vessels, but, as a rule, the convulsions are due to the irritant effect of the lesion. Later on epileptic or epileptoid convulsions are very commonly encountered. Sex plays no etiological part. Males and females are about equally affected. One-half the postnatal cases in children occur within the first three years of life.

Morbid Anatomy.—The post-mortem findings in these cases furnish various lesions. Sachs gives the following table:

Classification of Infantile Cerebral Palsies.

Groups.	Morbid Lesion.
I. Paralysis of intra uterine onset.	Large cerebral defects, porencephalia. Defective development of pyramidal tracts. Agenesis corticalis, highest nerve-elements involved.
II. Birth palsies.	Meningeal hemorrhage, rarely intracerebral hemorrhage. Later conditions: Meningo-encephalitis chronica, sclerosis and cysts, partial atrophies.
III. Acquired palsies.	Hemorrhage, meningeal and rarely cerebral. Thrombosis, from endarteritis, and in marantic conditions. Embolism. Later conditions: Atrophy, cysts, and diffuse and lobar sclerosis. Meningitis chronica. Hydrocephalus, seldom the sole cause. Primary encephalitis, the polio-encephalitis corticalis acuta of Strümpell. (?)

The difficulty of deciding the origin of the terminal conditions is very great and it is often impossible. Porencephalia, for instance, may be due to defective development, to embolism, to thrombosis, or to hemorrhage. The initial process of a diffuse sclerosis may be hemorrhagic or inflammatory.

Symptoms.—The clinical history of cerebral palsies in children varies in the three sets of cases. In the prenatal cases the condition is congenital, but may not be noticed for some time after birth. In birth palsies the condition is usually noticed shortly after birth or develops within a few weeks. In the later-acquired cases the patients may present an ordinary record up to the onset of the paralytic features. In their final development all present very common attributes and are usually indistinguishable by external examination alone. The great majority of natal and prenatal cases have bilateral palsies. After birth the tendency is to one-sided paralysis and after the age of three it is almost the invariable form. A pure monoplegia is so extremely uncommon as to be almost unknown.

Hemiplegic Cases.—The hemiplegic cases are the best type for study, as the unaffected side furnishes an opportunity for comparison. Ordinarily the paralytic features develop after an *acute febrile attack*, after or during an acute infectious fever, or in marantic states. The child is seized with *convulsions*, which are more severe on or entirely confined to one side of the body and to the side that is afterward paralytic. The convulsive attack is usually prolonged, lasting for several hours or even for a day or two, and perhaps returns several times within a few days. During the eclamptic seizures the temperature may be markedly elevated and more or less *unconsciousness* is commonly present. The child is left weak and exhausted, often with continued feverishness, and the loss of power on one side is frequently only incidentally noticed. If the child had formerly spoken, *speech*, as a rule, is temporarily lost, whether the paralysis be left- or right-sided, but after the age of about six years aphasia is well marked only in lesions of the left side in right-handed children. If their intellectual faculties are not destroyed, speech even then is regained with surprising rapidity.

The paralyzed limbs soon develop *marked spasticity* with *exaggerated reflexes*, which the spasticity may conceal unless the examiner be attentive to the play of tendons when the tests are made. The spastic condition is also followed by *contractures* which place the limbs in the positions so characteristic of hemiplegia in adults. Here also are similar *attitudes* and *gaits*.

Sensory disturbance is apparently absent even in the recent cases, and *electrical reaction modifications* are never present. The *trophic condition* of the paralytic limbs, however, is reduced, as is shown in the unequal growth on the two sides as time goes on. This results in some *deformity*. The shoulder girdle is smaller on the affected side, the chest and arm less in size, and the pelvis and lower extremity unequal to corresponding parts of the sound side. The half of the head and face on the paralytic side may be of inferior dimensions. The limping gait of hemiplegia is increased by the shortened limb, the pelvis tilts, the spine becomes scoliotic, and the retarded, paralytic, contractured upper member is held to the side of the body and usually flexed in all its joints.

From nine months to two years after the paralytic onset the paralyzed side, especially the hand, in over one-half of the cases, is animated by automatic and involuntary *choreoid* or *athetoid movements*, which often attain a wonderful complexity and range. They may also involve the face, but seldom to the degree that is observed in diplegic cases. These athetoid movements are usually intensified upon voluntary effort to use the limb. Attempts to grasp an object will often cause the fingers to move widely apart in extreme extension, and after clumsy, slow movements the object is awkwardly and insecurely held or the attempt fails. In some cases the extremities, particularly the upper one, are writhed about in the most vigorous, serpentine, and purposeless way, striking the patient's face or getting into awkward positions behind the neck or back.

The athetosis in rare cases is persistent day and night, in others it subsides during sleep, and in still others, and perhaps the majority of cases, it only appears when provoked by voluntary effort or emotional disturbance. In the cases that are marked by excessive athetoid motility the muscles, from constant though involuntary use, are frequently firm and well nourished. They may even be overdeveloped. Clark[1] has proven hypertrophy in such cases and actual enlargement of bones has been shown by the X-ray. On the other hand, well-marked atrophy may be encountered. The *joints* frequently present a marked increase in their range of motion so that the digits, for instance, can nearly be laid on the dorsal aspect of the hand. This condition is particularly marked in the severe cases, beginning very early in life. The ameboid and tentacle-like movement of the fingers and toes in athetosis, once recognized, can hardly be mistaken for anything else. *Associated movements* by which the paralytic member apes the positions and motions of the sound fellow reach their highest development in these cases in children.

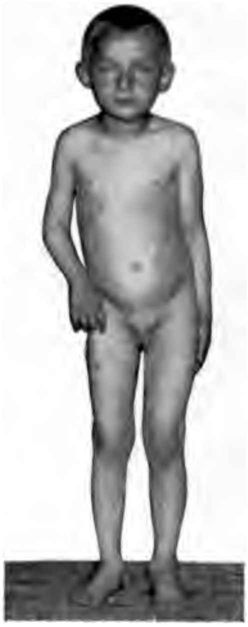

Fig. 90.—Attitude and deformity in cerebral right hemiplegia in a boy of seven.

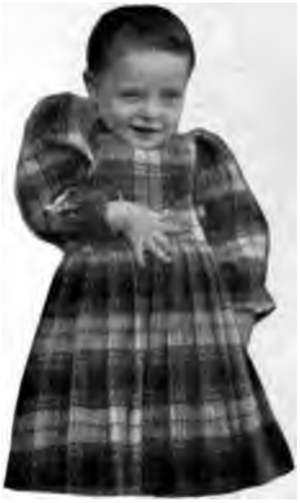

Fig. 91.—Right hemiplegia dating from birth, with overaction in right side of face and athetosis of right hand.

In the hemiplegic cases the arm usually recovers less than the leg or face and the athetoid condition is commonly confined to it. Contractures predominate in the foot and leg, producing an equinovarus, rarely a valgus, deformity, which is also favored by the shortness of the limb, and there is some tendency to contractured flexion at the knee and hip. The toes are frequently cramped and distorted by the contractures and hammer-toe is a usual deformity, but when athetosis is present an abnormal range of passive and active motion is found. In the face the tendency to contracture is frequently only shown in an overaction of the facial muscles during emotional expressions, as shown in figure 91, the boy being pleased at having his picture taken. Any degree of mental enfeeblement may be present in hemiplegic cases, but usually it is much less marked than in the bilateral form and may be practically absent. The tendency to *epileptic attacks* and mental deterioration is pronounced in the hemiplegic form, and will be mentioned again.

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[1] "Jour. Nerv. and Ment. Dis.," Nov., 1902.

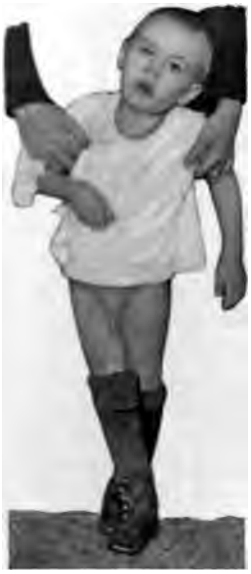

Fig. 92.—Diplegia dating from birth. Rigidities and spastic attitude with cross-legged gait.

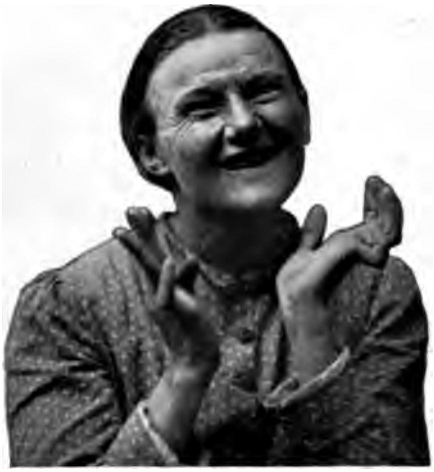

Fig. 93.—Diplegic congenital palsy with athetosis in face and all extremities.

Diplegic Cases.—In the bilateral form the unilateral conditions of the hemiplegic cases are present on both sides, but the lower limbs are, as a rule, more strongly affected than the upper and the face may show little or no motor impairment. In rare cases the trouble seems to be limited to the lower limbs, and these instances are sometimes described as *paraplegic*. Almost invariably, however, the presence of speech difficulties, of mental defect, and of clumsiness in the use of the hands will betray the diplegia. The frequency of spinal hemorrhages in stillborn children, as determined by Spencer, for instance, gives ground to suppose that in very exceptional instances the cord-lesion may alone occur and a true paraplegia result if the child survive. These bilateral cases are mainly of prenatal and birth origin, only a small number arising after the first few months of infancy and practically none after the third year of life. The more extensive injury to the brain is attended almost of necessity by greater mental impairment and idiocy marks many cases. Microcephalia sometimes results or the skull may have so defective a shape that porencephalia and anencephalia may be suspected. Such children are either noticed to be rigid and inactive at birth, or after birth-injuries and convulsive manifestation develop rigidity and contractures during the early months of infancy. Frequently it is only when the child is found incapable of learning to sit up or to use its legs in efforts at walking that the paralytic state is recognized. Mental backwardness, slowness in the development of speech, and other indications of injury to the highest brain functions are often neglected until the third or fourth year in the misguided hopefulness that the child will "outgrow it." The rigidity and spastic state is frequently so great that the legs and arms present a "lead pipe" resistance to passive movements. Voluntary efforts are hindered or defeated, and some of these children never get the hands to the face, much less their toes to their mouths. They are difficult to handle and to dress. Their arms and legs are as unmanageable as stiff-jointed manikins. They are "all thumbs" and awkwardness. The spasticity in the lower extremities, which tends to flex hips and knees and especially to adduct the thighs, holds the knees closely together, makes it difficult to dress and bathe them as infants and defeats locomotion later on. Placed on a chair, the lower limbs have a tendency to maintain a rigid horizontal position. If they are placed on their feet the legs cross, the heels can not be brought to the floor, and if steps are taken it is only by advancing the foot that is in front and then bringing up the one in the rear. As they grow older a tendency to equinovalgus or varus and genu valgum is induced, but by keeping the feet widely separated they are able to dodge one knee around the other. They thus advance in a shuffling, knee-rubbing, toe-scraping, laborious manner, to which the increased reflexes and ankle-clonus give a jerky, tremulous, spastic character.

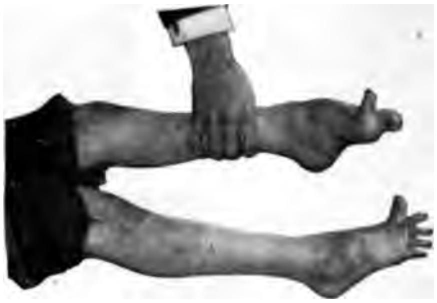
Fig. 94.—Athetosis of feet.

In some cases more or less athetosis is present in all four members, and occasionally it invades the face and even the tongue, pharynx, and larynx. When the athetosis is general, and particularly when it involves the feet, the patients are rendered almost completely helpless physically.

Little's Disease.—English, German, and American writers are prone to use the term Little's disease in a generic sense to embrace all cerebral palsies in childhood. French writers, of the Salpêtrière school especially, insist upon a clinical form of spastic diplegia to which they give the distinctive name of Little's disease, after the London surgeon who among the first called specific attention to this class of disorders. As these cases referred to present certain definite characters and a more hopeful future than the others, they merit separate mention.

The initial condition is a premature birth or the birth at term of a markedly undeveloped child. This means a defective pyramidal tract in the spinal cord and brain. Such children usually weigh less than four pounds. Spasticity results in the territory of the lower neuron from the lack of control due to the undeveloped condition of the upper neuron. These children are rigid from birth, but the mental qualities are not necessarily diminished, and if they survive, the tendency is to continuous improvement as the pyramidal tracts develop. This development is never complete, but continues up to the age of full growth. The motor condition is one of pure spasticity and there is no tendency to athetoid movements or epileptic attacks. The gait remains more or less spastic through life, which may be a long one marked by ordinary or even brilliant mental activity.

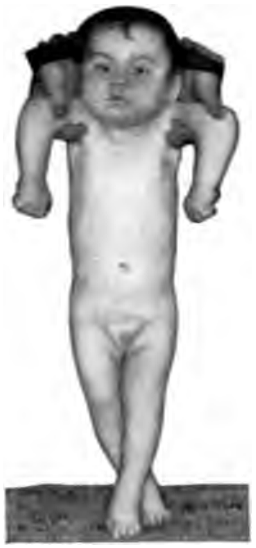

Fig. 95.—Case of Little's disease. Rigidities and cross-leg from spasticity in a child weighing three pounds at birth.

Amaurotic Family Idiocy.—Sachs[1] proposes this clinical designation for a group of cases of which he collected nineteen occurring in ten families, with three autopsies. Numerous other observations have since appeared in current literature, notably those reported by Collier.[2] These children are born at term and present healthy physical and mental development up to the age of four to ten months, when they become weak, lethargic, and stupid. Ocular symptoms soon appear and blindness due to optic atrophy develops. Frey [3] does not agree with Sachs that the condition is merely agenetic, but considers the changes found to be postpartum, presenting for childhood the state recognized as amyotrophic lateral sclerosis in adults. The ophthalmoscopic picture is said to be very characteristic. Beard[4] states that the appearance is not that of pronounced atrophy. The disc is not markedly blanched, but the fovea centralis presents a clear-cut liver-colored plaque surrounded by a halo of grayish-white which does not obscure the retinal vessels. Nystagmus, ocular deviations, and pupillary anomalies are frequently encountered. Autopsical findings have shown convolutional simplicity and cortical cellular degeneration. Sachs [5] reports degenerative changes in the gray matter of the entire cerebrospinal axis and even in the root ganglia. These changes have been attributed to toxic processes, but none of the alleged toxic factors can be accepted as competent in all instances. As many as four cases have been observed in a single family, and the inherent anatomical lack in the brain is not explained by inherited syphilis, which may, however, produce a very similar clinical picture.

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[1] "New York Medical Journal," May 30, 1896.

[2] "Brain," 1899.

[3] "Deut. Arch. f. klin. Med.," Bd. lxviii.

[4] "Jour. Nerv. and Ment. Dis.," May, 1900.

[5] "Jour. Nerv. and Ment. Dis.," Jan., 1903.

Epileptic Attacks.—One of the most serious features of the cerebral palsies of children is the marked tendency to the development of *epileptic attacks*. These appear in fully one-half of all cases. They are due to the brain-lesion, and usually are most marked in the paralytic limbs, but with a tendency to spread and become generalized. True Jacksonian fits are but rarely encountered. In some cases the initial eclamptic attack is at once followed at regular intervals of weeks or months by these seizures. In others they do not appear for a period of years and may be preceded by petit mal attacks. They are always to be expected, and when established constitute a serious menace to the health and mental state of the patient. The ordinary tendency of epilepsy to induce dementia is exaggerated in these already defective brains.

Diagnosis.—The diagnosis of cerebral palsy when the condition is well marked should present no difficulty. In the slighter grades the epileptic attacks or the choreoid movements are frequently mistaken and the organic basis overlooked. The combination of head symptoms and mental enfeeblement with the motor difficulties in diplegic cases should be sufficiently striking. The hemiplegic distribution of paralysis can tell but one story. In cases presenting paraplegic features it may require a careful investigation to locate the trouble in the brain. The history here, as elsewhere, is a valuable guide. In addition the preserved electrical reactions, the comparatively normal trophic conditions, and the exaggerated reflexes should readily exclude a cord-lesion. The teratological defect which furnishes the cases of amaurotic family idiocy and the defective development of Little's disease must both be deciphered from the clinical data or the post-mortem findings.

Prognosis.—The prospects in these diverse cases vary widely, but it may be positively stated that complete recovery never occurs and that a perfectly normal condition is never attained. In the cases of prenatal origin the prognosis must rest on the observed course of development during the first year of life. Improvement in motor control and the appearance of mental awakening are hopeful indications, but the majority of these cases are doomed to mental retardation or idiocy and more or less physical helplessness. The cases that conform to the type of Little's disease are exceptions. In these the prognosis for mental development is good and progressive motor improvement, up to a certain point, may be confidently expected. Amaurotic family idiocy, which is a congenital defect, is, at present, of absolutely fatal prognosis.

In the cases of birth palsy the intensity of the convulsions, their persistence, and the apparent extent of brain injury, in a very rough way, guide the prognosis as to life. If, after a few months, spasticity is still absent and some voluntary motion occurs in the paralyzed limbs, the prospects are much improved. The question of mental development can only be decided by watching the course of events. In the acquired cases the prognosis is much the same as in the apoplexies of adults as far as motility is concerned. The return of motion in the paralytic limb and the absence of contractures permit hope of a fair degree of motor restoration. Speech is usually restored, unless the frontal lobes have been injured and mentality impaired. The loss of sight and more especially of hearing is likely to prevent mental development and to induce idiocy. Whenever epileptoid attacks appear, they constitute a very serious feature and foreshadow dementia. They are somewhat more common in the late than in the congenital cases and in the hemiplegic than in the diplegic palsies.

Treatment.—In the birth-palsy cases the nutrition of the child is the first problem, as suckling and swallowing are often impossible. The evidence of meningeal hemorrhage is usually so indefinite that trephining, which has been proposed, can rarely, if ever, be practised. The convulsions must be subdued by cold applications to the neck and head, and by sedatives, such as chloral, bromid, morphin, and by continued chloroformization, if necessary. The bowels should be thoroughly opened, preferably by means of calomel. The same measures are indicated as in older cases of acute hemiplegic convulsions and in convulsions generally.

In the later cases the management of the apoplectic and paralytic states is the same as for adults (see p. 214). From the first, the nutrition of the paralytic muscles and the tendency to contractures must be met by massage, passive movements, and faradism. As soon as contractures appear, the massage and electricity must be used only on the weaker muscles—that is, as a rule, only on the extensors. The use of these measures to increase the strength of the overacting muscles may occasion decided harm. Unfavorable positions of the limbs must be obviated by mechanical measures, if necessary. Such means may do much to retard contracture, to hasten and increase voluntary control, and to favor growth in the paralytic members. Orthopedic appliances and tenotomies in neglected cases sometimes secure much permanent improvement. Transplantation of tendons has lately come into prominence, and in some instances can be resorted to with fair prospects of improvement.

Athetosis may be subdued to a considerable extent by fixation appliances or plaster-of-Paris splints. Hammond reported some cases temporarily much benefited by nerve-stretching, and Horsley has extinguished the athetosis by excising the corresponding brain-centers. In one such case the writer advised the removal of the cortical centers for a very unruly upper extremity, and the resulting palsy in the member was replaced finally by slight but useful voluntary control.

When idiocy is present it is open to educational methods only. The epilepsy that so frequently complicates these cases can be controlled to some extent by bromids or combinations of various sedatives with bromids. When the attacks are of a distinctly Jacksonian character, the question of operation will come forward. Every case must be considered by itself. As in many instances the condition is one of cerebral sclerosis, no operation can do good. If there be a cystic state, exploration and evacuation may be productive of much relief. In some cases the mere effect of the operation has been temporarily beneficial; in others it has acted by relieving pressure. Removal of cortical centers in Jacksonian fits of this class is very likely to merely displace the initial symptoms and to add to the traumatic conditions already present. Linear craniotomy when the brain is diseased can only be condemned. If it have any place it is in the cases that present closed fontanels at birth with microcephalic heads, the possible result of early synostosis.

CHAPTER XI.

TUMORS OF THE BRAIN.

THE encephalon is frequently invaded by various new growths common to other parts of the body, and by a number of neoplasms that are practically found only within the skull. In addition, new formations arising from the meninges and cranial walls, while not strictly brain-tumors, present symptoms that are identical with lesions of the cortex, and are localized in the same way. The term "brain-tumor" is here taken broadly to cover new formations within the skull.

Etiology.—The causation of brain-tumors is an obscure subject. Many of them arise by *metastasis* from distant morbid fields, as is usually, if not always, the case in tubercular growths, carcinomata, and parasitic cysts and masses. Some arise by intracranial extension from the orbit, pharynx, and cranial bones. *Heredity*, aside from tubercular cases, plays a very insignificant if not an entirely negative rôle. *Traumatism* has probably been too frequently assigned as the inciting cause of brain-tumor, but it undoubtedly is competent in some instances. Thus, continuous symptoms have arisen within a few days of a head injury, and a tumor has subsequently been found at the traumatic site or in the brain beneath. There can be no doubt that even slight head injuries may localize tubercular and syphilitic activity and lead to corresponding tumor growth. Sarcoma and glioma are the neoplasms that seem to be most frequently attributed to trauma, but at the same time they are among the most common of brain-tumors.

No *age* is exempt from tumor growth within the cranium. The great majority of cases occur in childhood and active adult life. The activity of tuberculosis in childhood, and the prevalence of syphilis in young adults, as well as liability to traumatic influences, may in part account for this. *Males* are somewhat more subject to cerebral tumor than *females*.

Pathological Anatomy.—Of all brain-tumors tubercle furnishes the largest proportion. The implantation of the tubercular process within the brain sometimes gives rise to the formation of large, solitary, isolated, often encysted caseous masses that may be *single*, but are *multiple* in about one-half of the cases. Tubercular tumors favor the base and the course of the large cerebral and cerebellar vessels, which recalls the usual propagation of tubercular meningitis. No part of the brain, however, is exempt. They do not destructively invade the brain-tissue, but displace it and act as foreign bodies, causing pressure atrophy. About them may often be found an area of tubercular infiltration and inflammatory activity, especially if they are seated so as to involve the meninges. Three-fourths of the cases occur before the age of twenty. The tumors vary in size from a pea to a hen's egg, or even a larger size, and in number from one to a score.

Sarcomatous growths are next in frequency. Usually they present the distinctive and important character of being more or less encapsulated and sharply separated from the brain-tissue, from which they can, therefore, if accessible, be easily enucleated. As a rule, they are outgrowths of the cranial dura or periosteum, and the majority of them are situated in the basilar region, involving the brain-axis. In comparatively rare instances they infiltrate the brain-tissue, or, arising at the vertex, spread out broadly. They are of rapid growth, and in a given case furnish pronounced, persistent, and uniform symptoms. They present the variations of cellular elements that mark sarcomata elsewhere. As a rule, they are single.

Glioma is peculiar to nervous structures. It arises from the neuroglia and finds its most usual seat in the brain, though it may occur in the spinal cord or retina. It presents a reddish, vascular color and a soft consistency quite like that of the brain-substance. Glioma is eminently an infiltrating and a destructive growth. Its outlines are difficult to determine. Owing to its strangulating effect, the center of a glioma is frequently filled with softened detritus and fatty-degeneration products, which may become fluid and the growth thereby cystic. Many alleged local "brain-hypertrophies" have been gliomatous infiltration in fact, and the microscopic examination of "cyst-walls" has alone discovered the true nature of the lesion in other instances. The favorite location of glioma is the white substance near the cortex, and it may infiltrate large areas, even the greater part of a hemisphere. Ordinarily it is single.

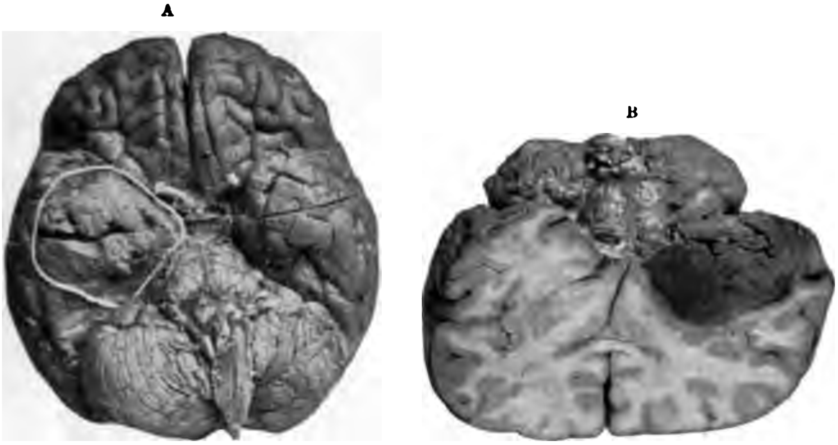

Fig. 96.—**Sarcoma** of right temporosphenoidal lobe. A, Basilar surface; B, transverse section (Dr. W. A. Jones).

In consistency they are soft, often highly vascular, and may in some cases be easily mistaken for angiomata. Hemorrhages not infrequently occur in them. In other cases a great amount of interstitial fluid gives them a myxoid character.

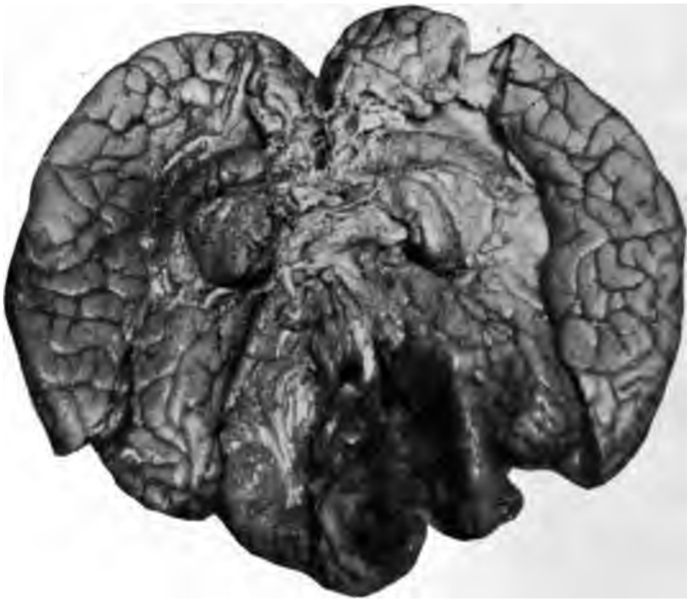

Fig. 97.—Glioma of cerebellum containing recent and old hemorrhages.

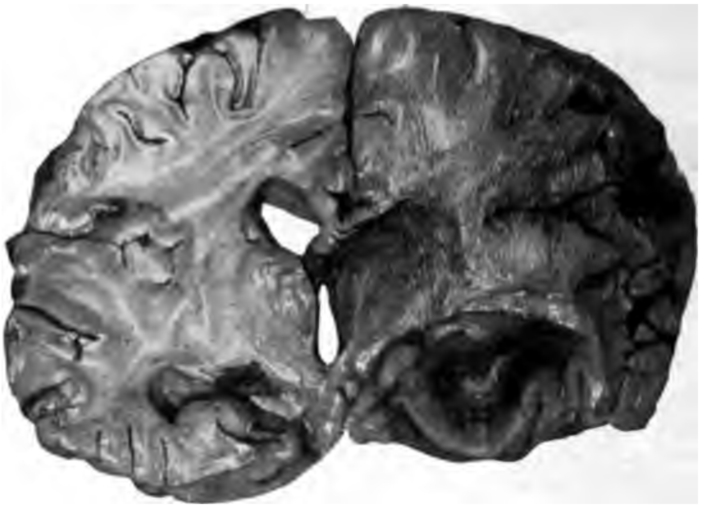

Fig. 98.—Gliomata of right temporosphenoidal lobe.

Carcinomata, practically speaking, are found in the brain only as metastatic growths arising from primary cancer in the body organs, breast, etc., or through direct invasion by extension from orbital or other cranial and facial carcinomatous growths. They are also practically confined to the second half of life. Uncontrolled by the soft tissue of the encephalon, carcinomata rapidly invade the brain, forming ill-defined, nodular, rarely encapsulated, very vascular, and practically inoperable tumors. When arising secondarily from cancer in the body organs, especially those in the thorax, carcinoma of the brain locates by preference in the neighborhood of the great vessels of the base. In other instances it springs from the choroid structures and the epithelial lining of the ventricles, or from the dura and pia mater.

Cysts form tumors in the brain with comparative frequency. An encysted hemorrhage or softened infarct does not ordinarily give rise to progressive or marked irritative symptoms, and need not be here considered. An encysted and latent abscess may, however, produce all the symptoms of a brain-tumor, which in fact it is. Cysts arising from parasites, such as the cysticercus and echinococcus, are rather common in some parts of the world, but are distinctly rare in the United States. Diamond[1] could only find eight reported cases of cerebral cysticercus in American literature. The central breaking down of sarcomata, and especially of gliomata, produces cysts the nature of which may be indeterminate without histological search. Rarer forms are due to ependymal inclusions in embryonic development, producing later in life ventricular cysts or cysts in the posterior portion of the pituitary body. Dermoid cysts have also been encountered within and outside the dura, and especially in the cerebellum. Cyst development is usually slow, and the resulting tumor acts as a displacing foreign body. Their diagnosis is important, as they are readily evacuated and the cyst-wall may be removed when situated in a surgically approachable part of the brain.

Syphilitic tumors of a gummatous sort in the brain are more frequent than statistics would indicate. Owing to their partial amenability to treatment and their readily accepted consequences they are seldom reported. As a rule, they spring from the meninges and large vessels, and, when deeply seated, are usually the ingrowth from one of the penetrating folds or vessels. They may be definitely limited or surrounded by softened brain-tissue, and have no tendency to infiltrate. Their usual location is in the hemispheres, especially at the base, or in the pons. They rarely occur in the cerebellum or central ganglia. After treatment their early soft, caseous, and gelatinous character may be changed to a shrunken, degenerated, and fibrous condition. They are usually multiple, nodular in form, and about the size of a chestnut. They occur as the result of acquired syphilis, and consequently usually in adults at periods varying from a few months to many years after the primary lesion. They are commonly attended by other syphilitic disturbances of the brain, such as endarteritis, meningitis, and cranial-nerve lesions. Of rapid growth, they usually promptly recede to some degree under adequate treatment, but their absolute removal by medication is always questionable.

Many other tumor forms are occasionally found in the brain. *Cerebroma* is an embryological infolding of the gray mantle, which becomes occluded, usually in the white substance, and, later, takes on active proliferation, forming a heterotopic tumor. *Fibroma, angioma, neuroma, psammoma, papilloma* of the choroid plexus and Pacchionian bodies, *actinomycosis, lipoma, teratoma, osteoma, cholesteatoma,* etc., merely require mention. The *pituitary tumor* associated with acromegaly is discussed under that head, but tumors of various sorts, such as adenoma and adenosarçoma, occur at this point without such association.

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[1] "Jour. Am. Med. Assoc.," June, 1899

Aneurysms sometimes give rise to symptoms of intracranial tumor. They arise, especially after middle life, as the result of arterial disease, discussed in chapter v, or they may be due to traumatism. Aneurysms of a cirsoid character and of great extent that produce brain symptoms are occasionally encountered on the meningeal arteries. The cerebral vessels present aneurysms usually of a globular or pyriform shape. Their favorite location is at the base and in the Sylvian fissures, at the bend of the carotids and in the basilar artery, but they occasionally develop on the perforating arteries and may attain considerable size. The formation of *miliary aneurysms* and their relation to cerebral hemorrhage have already been indicated. Occasionally a patient with cerebral aneurysm is conscious of its pulsations and hears the bruit. The bruit may also sometimes be heard by auscultation of the cranium, but vascular murmurs have also been heard in a case of extensive softening. The chief symptoms of aneurysm are due to pressure upon the cranial nerves and brain-centers.

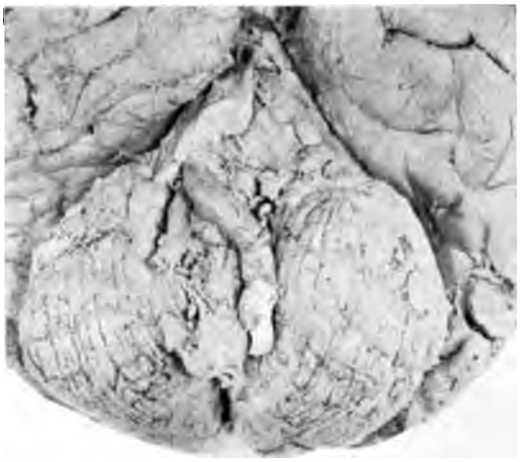
Fig. 99.—Aneurysm of the basilar artery.

All brain-tumors cause more or less circumferential destruction of brain-tissue. This is at a minimum in slow-growing tubercles and reaches its maximum in rapidly growing and infiltrating glioma. Almost invariably, if the meninges are reached, an inflammatory thickening over the growth is added. In the case of tubercle and syphilis a more or less diffuse meningitis is commonly present, sooner or later. The interpolation of a new growth within the resisting walls of the skull means increased intracranial pressure and disturbed vascular conditions. The effects are serious, relative to the intensity of the pressure, the rapidity with which it is established, and the location of the growth. Even a small tumor in the circumscribed space under the tentorium produces pressure rapidly. In the same way, if located near the falx or other resisting structure, its pressure effects seem to be greater. On the other hand, if of slow growth and of a non-destructive character, a tumor may displace large portions of a hemisphere, especially in the latent regions of the right side, and attain enormous proportions, without producing notable symptoms. This pressure is felt not only in the vicinity of the tumor, but throughout the encephalon, and may cause symptoms and even inflammatory disturbance at a distance. Tumors so located as to obstruct the return circulation from the ventricles by occluding the straight sinus and the veins of Galen produce a dropsical condition of these cavities, an internal hydrocephalus, with extreme pressure indications. Even the bony walls of the cranium over the tumor are thinned and eroded in some instances, and that, too, when the tumor is not superficial.

A number of observers have noted changes in the *spinal cord* in brain-tumor cases. These seem mainly to affect the posterior portions of the cord and the posterior roots. Ursini[1] considers them of toxic character, and Lubarsch has found somewhat similar changes in a case of gastric cancer. The analogous changes in anemias and cachexias are significant in this connection. Kirschgässer,[2] however, believes the cord changes to be secondary to increased tension in the dural sheath. Batten and Collier[3] take a similar view, and find such changes in over half of all cases. Certain cases in which the tumor disintegrates the motor tract within the skull show degenerative changes in the direct and crossed pyramidal tracts in the cord.

Symptoms.—The symptomatology of brain-tumors presents the widest variations. The clinical picture is dependent upon the situation of the growth and modified in its evolution by the nature of the tumor. A slow-growing mass starting from the meninges may deform an entire hemisphere without giving rise to symptoms, while another of insignificant volume may produce the most marked motor, sensory, and mental disturbances, or lead to sudden death. We may divide the symptoms of brain-tumor into: (1) Those which are presented irrespective of the nature and location of the growth,—*general symptoms;* (2) those dependent upon the situation of the neoplasm,—*focal symptoms;* and (3) *topical symptoms*, those at the superficial site. Under certain conditions the general symptoms have some localizing value, as will be pointed out *seriatim*.

General symptoms of intracranial growths are: (1) Headache; (2) generalized convulsions; (3) mental impairment; (4) double optic neuritis or optic atrophy; (5) vomiting, and (6) vertigo. Temperature changes above and below the normal, alterations in respiration and pulse rhythm, polyuria, glycosuria, insomnia, delirium, stupor, coma, slow speech, and malnutrition are common to brain-tumor and all other encephalic lesions, especially in their terminal stages.

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[1] "Deutsch. Zeit. für Nervenh.," 1897.

[2] "Deutsch. Zeit. für Nervenh.," Bd. 13.

[3] "Brain," 1899.

Headache.—In the great majority of cases of intracranial tumor headache is an early and persistent symptom. It has no necessary relation to the location of the tumor, and is usually frontal or occipital, less frequently parietal, or at the vertex. Sometimes the eyeball is the seat of pain. It usually increases with the increase in the tumor's size, and is aggravated by any effort or condition which increases arterial tension and cephalic hyperemia. In character the headache is dull, heavy, persistent, often with great exacerbations; frequently it prevents sleep, and sometimes is of an intensity that becomes intolerable and submerges the patient's intellect. It has even been considered the cause of death. In rare instances a persistent, circumscribed headache has been found to conform to the tumor's location, and, therefore, like all diffuse symptoms, it may exceptionally have a localizing value. In such case there is often local tenderness on percussion, and sometimes local elevation of temperature. In cerebellar growths local pain and pain in the neck, with retraction of the head, are significantly common, but a cerebellar tumor may occasion continuous frontal pain. Pain in the distribution of the fifth cranial pair, or in a single branch, is an indication of local irritation of the nerve that may be confounded with the diffuse headache.

General Convulsions.—Over one-half of all cases have general convulsions at some period of the disease. They may be the early and only manifestations of the tumor, and indistinguishable from ordinary epilepsy, for which these patients are not infrequently treated during long periods of time. More often the convulsions are preceded by headaches and focal symptoms. As a rule, Jacksonian fits eventuate in the generalized form, either by gradation or alternation. While these commonly conform to the clinical type of the ordinary epileptic seizure, a careful study of them sometimes enables one to detect variations from the type that have some significance. The onset is less violent and abrupt. The clonic stage is likely to be protracted to fifteen minutes, a half hour, or longer; there is less depth to the coma, and the subsequent deep sleep may be much abridged or entirely wanting. They rarely appear with the regularity that is common in ordinary epilepsy. Attacks of the *petit mal* variety are rare in brain-tumor, but do occur, while epilepsy presenting major attacks is rarely without minor manifestations.

Mental Impairment.—The final stage of tumor cases is customarily marked by great hebetude and a stuporous condition that may increase into final coma. In lesser degree this belittling of mental vigor is common in the earlier periods of the disease, with or without convulsions. When the headache is intense, the patient is likely to seek seclusion, hold his head in his hands, and present a picture of dejected indifference that clearly indicates his mental state. He replies slowly to questions or disregards them utterly. He can not think quickly, and is sluggish in all his mental qualities as well as in his physical attributes. The pulse decreases in strength and rapidity, respiration is slow and shallow, and if suffering ceases the patient sinks into a sort of hibernating sleep. From this apathy, which may be more or less marked, he partially rouses, or he may decline into a more comatose state and die of inanition. The two prominent characteristics of the mental state are apathy and suffering, which may be associated or alternate. So far as mental action can be elicited it is usually unclouded, except in rare periods of delirium. Something of this apathy may be noticed in comparatively recent cases, and, as a rule, brain-tumor patients are little worried over their condition. This mental change is most likely to be well marked, and to appear early in tumors of the frontal lobes. Gianelli[1] reports 77 cases of tumor in the frontal lobes accompanied by mental disturbance and 20 in which morbid psychic phenomena were lacking. In rare cases the mental symptoms closely imitate the formulated insanities, such as mania, melancholia, and even hallucinatory paranoia.[2]

Optic neuritis, or papillitis, occurs in about eighty per cent. of encephalic growths. In very rare instances only one nerve-head, usually on the same side as the tumor, has been found affected. In many cases the condition is more marked in one eye than in the other, and there is some reason to think that the more intense inflammation is also usually on the same side as the lesion. (For further details regarding this lesion of the second cranial nerve reference is made to Part II.) In chronic cases papillitis may suddenly appear, but a chronic papillitis or a very insidious one does not belong to an acute or rapidly developing lesion. The natural result of choked disc is *atrophy* of the optic nerve, which, therefore, has the same significance in the presence of other tumor indications. It must be borne in mind that central vision may not be greatly disturbed when the discs are distinctly choked, and their examination should never be neglected. This general symptom of brain-tumor is very constantly found in growths involving the cerebellum, geniculate bodies, corpora quadrigemina and the brain-axis. It, therefore, has a slight localizing value.

Vomiting.—Attacks of vomiting, usually of the so-called cerebral variety, without fermentation, nausea, and effort, are common. They may last for a few days or weeks; subside and recur, and they usually attend other indications of tumor extension or invasion. Vomiting sometimes threatens death by inanition. Like choked-disc, it is most frequent in tumors involving the cerebellum, especially the middle lobe and the corpora quadrigemina.

Vertigo occurs with brain-tumors in all locations, but especially in the cerebellar and frontal regions.[3] It is a vague distress in some cases or a feeling of being submerged or of mere darkness and sinking, and may be paroxysmal or constant. In addition, brain-tumor may give rise to ocular vertigo by involvement of the motor oculi nerves, and to auditory vertigo by implicating the eighth nerve. In cerebellar tumors affecting its peduncles, especially the middle one, there may be forced movements and attitudes. In these combinations vertigo furnishes a valuable localizing indication. In some cases it is an epileptic equivalent. It sometimes is attended by vomiting. It may prevent the patient's rising from recumbency, or cause him to lie down if standing. Ordinarily he grasps an object for support, and is soon over the attack.

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[1] "Il Policlinico," July 15, 1897.

[2] Bayerthal, "Münch. med. Wochens.," 1899.

[3] Bruns, "Wien. klin. Runds.," 1897, No. 46.

Focal Symptoms.—The symptoms due to the presence of a new growth or other lesion in special brain regions have been discussed at length in the opening chapters of this part. They consist usually of unilateral *spasms* confined to the face, to a limb, or to a segment of a limb; of *monoplegias;* of *paresthesias* of functional distribution; of *hemianopsia* or other sensory disturbances; of *aphasia;* of *stereoagnosis* and of impairment of the *cranial nerves.* They are variously combined as associated anatomical structures happen to be involved. They advance with the tumor's growth, and change as irritation yields to destruction. They thus vary indefinitely in different cases, and usually present numerous modifications in the clinical history of any given case. Williamson[1] gives particular importance to the significance of hemiplegia of very gradual onset which may at first be entirely unattended by the other usual symptoms of brain-tumor. To the Jacksonian fit, whether the spasmodic or the sensory features predominate, most attention has been directed, as it points with definiteness to the locus of greatest and usually of initial disturbance. To avoid needless repetition, the reader is again referred to the previous chapters on cerebral localization and diseases of the cranial nerves. *Hallucinations* of the special senses occurring with convulsions or independently sometimes are caused by tumors situated in the corresponding sensory cortical area or influencing it more or less directly.

Topical Symptoms.—The location of a new growth near the surface of the brain, so that it impinges upon the meninges or invades them, is sometimes attended by topical conditions of some significance. *Local and persistent tenderness, heat, and headache* may be thus induced. In the rare cases, where the cranial bones are perforated by erosion, *palpation* may detect the bony opening and the tumor mass. Invasions of the orbit, pharynx, and nasal cavities are open to ready investigation. *Percussion* over large tumors superficially placed may demonstrate a changed pitch in the resulting note.[2]

Multiple tumors may furnish many diverse and conflicting symptoms, but usually one, owing either to its situation or major size, gives a preponderance of manifestations.

Course.—The majority of cases of brain-tumor are clinically of insidious onset, progressive development, and fatal termination in a cachectic state, induced by the gradually increasing intracranial pressure and the extinction of cerebral functions. The ordinary duration is from a few months to three years. The lethargy, stupor, and coma of late stages are frequently aggravated by paralytic features, and continuous vomiting may defeat all attempts at nourishment. While this is true of the larger number of cases, there are numerous exceptions. Sometimes the first pronounced symptom is an apoplectic attack which may carry off the patient at once. The apoplexy is brought about by a cerebral hemorrhage, due to erosion of a vessel, or sometimes to the tumor pressure obliterating an artery by thrombosis or by its mere mechanical effect. In the softer growths, notably gliomata, hemorrhage into the mass may take place and secondarily affect the cerebral structure. Hemorrhage into the substance of a tumor may also directly induce the apoplectic state and lead to a fatal termination. Gerhardt states that in this accident the initial fall of temperature that attends an ordinary cerebral hemorrhage does not occur. Some tumors give rise to no symptoms and are only detected after death from intercurrent disease.

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[1] "Practitioner," Sept., 1904. [2] Bruns, "Wien. klin. Runds.," 1897, No. 46.

The nature of the growth in some degree determines the rapidity of the evolution of the case, but in turn is subject to its localization. Tubercle, fibroma, cerebroma, sarcoma, syphiloma, carcinoma, and glioma, in ascending order, increase in the rapidity with which they influence the brain-structure. The more rapid the growth, the more rapid the development of pressure and the more quickly does destruction advance. Tubercles, however, furnish some of the most rapid as well as some of the most dilatory cases. A fatality is imminent in proportion as the growth recedes from the cortex and approaches the medulla in location. In the latter position or in its neighborhood, as in the basal ganglia, the pons and cerebellum, a small and comparatively benign growth may induce a rapid course and early fatal termination.

The succession of symptoms is again determined by the nature and the location of the growth. Headache is, of all the diffuse or general symptoms, commonly the earliest, and optic neuritis usually follows in a few months. Spasms depend on the amount of irritation of the cortical structures, and paralysis on their destruction or the cutting of the motor paths. General convulsions, as pointed out, are often early features; they especially precede the paresthesias and palsies. By the extension of the growth invasion symptoms are set up (see p. 188), and a widening of motor and paralytic phenomena follows in anatomical order. Tubercular and carcinomatous growths, being usually secondary manifestations, bear with them the possibility of a rapid downward course from conditions outside the cranium. In addition, a tubercular neoplasm is likely to set up a diffuse tubercular meningeal infection that may promptly destroy the patient. A syphiloma in the same way may be attended by a wide-spread syphilitic meningitis.

Diagnosis.—In a case presenting cerebral indications and giving rise to the suspicion of tumor, the diagnosis is much facilitated by a careful arrangement of the symptoms in the order of their development. A number of problems are presented: (1) Is there a tumor? (2) Where is it located? (3) What is its size? (4) What is its nature? (5) Is it operable?

To the solution of the first question, is there a tumor, a definite answer can usually be given. Bruns says tumor may be diagnosed in eighty per cent. of all cases. After taking into consideration the evolution of the case, the focal or localizing symptoms are the most reliable data, but are strongly confirmed or rendered positive by the presence of the diffuse indications. Localized fits may be presented by ordinary epilepsy, but if headache, cerebral vomiting, vertigo, and choked disc, one or all, are added, the probability of tumor is enhanced. Should now the local spasm be followed by persistent paresis or paresthesia in the same locality, a positive statement is allowable. In the same way a hemianopsia or aphasia, following or associated with generalized or limited convulsions and attended by some or all of the diffuse symptoms, permits a diagnosis of tumor.

The clinical history of the case is important, as by it and its special symptoms we have to differentiate tumor from acute and chronic *meningitis*, with which, indeed, it may in some cases be complicated. The early and persistent headache is common to both, but tumor presents, as a rule, a choked disc, while meningitis has a neuroretinitis. Meningitis, even of the tubercular form, is of comparatively rapid development, while tumor commonly requires months. In meningitis the motor symptoms are usually bilateral, as contrasted with the one-sidedness of tumor disturbance. It will be recalled that a latent *cerebral abscess* may present all the indications of a tumor, which indeed it is, and it requires no differentiation aside from the fact that it offers a better operative outlook. The *cerebral palsies* of childhood and *general paresis* in adults have been confounded with tumor, but a careful scrutiny of the case, with a knowledge and mindfulness of these maladies, will obviate error. The tumors which present a sudden apoplectic onset may be confounded with *cerebral hemorrhage* or softening. Here the ordinary antecedents of vascular disease and the arterial condition presented furnish a strong presumption of vascular accident, and serve to a reasonable degree to exclude tumor, if the diffuse symptoms of tumor are lacking. It will, however, be borne in mind that a brain-tumor may bring about vascular disease, and that hemorrhage into gliomatous and other soft growths may produce apoplectic states which would be further favored by atheromatous conditions of the blood-vessels. The diagnosis must rest on the antecedent and sometimes on the subsequent history of the case.

There is reason to hope that *skiagraphy* may give aid in the diagnosis of brain tumors. Obici and Ballici [1] were able to thus demonstrate a sarcoma in a cadaver. The writer,[2] in two cases, and Mills,[3] in two cases, have obtained similar results during life.

The second question, of *location*, is answered, if answerable at all, by a consideration of the focal and topical symptoms of the case and the localizing bearing of the diffuse symptoms that are presented. The absence of focal symptoms points to the regions of latent lesions in the frontal, temporosphenoidal, and postparietal regions, especially on the right side.

It is necessary to answer the second question before the third can be approached, as we can only relatively estimate *the size* of a tumor by knowing its location and the anatomical regions invaded. To this end the sequence of developments is our greatest aid. Invasion symptoms enable us to trace, especially in the motor cortex, the gradual growth of a tumor, and in some directions to indicate its extent. If this can be done on a portion of the periphery, a knowledge of the symptoms that would arise by the tumor's extension in the opposite

[1] "Rivista di Pathologica," Oct., 1897. [2] "Amer. Jour. Med. Sciences," Feb., 1899.
[3] "Phila. Med. Jour.," Feb. 24, 1902.

direction may enable us to say whether or not such centers or pathways have been seriously invaded. It is evident that only a very inexact measurement can be made, and this is especially true if the growth is situated in the neighborhood of the latent lesion territories.

To the fourth question, what is *the nature* of the tumor, a positive answer can never be given, unless there is an external portion of the tumor, but a strong probability is frequently forthcoming. In this relation the age, the diathesis, the history of previous illness, the presence of various diseases, the location of the tumor, its rate of development, and the effect of treatment aid us. In childhood, tubercle, glioma, sarcoma are most frequent; in adults, the firmer tumors and syphiloma; in advanced years, carcinomata. The presence of tuberculosis, carcinoma, parasitic cysts, or syphilis gives much weight to the supposition that the brain-lesion is of a similar character. Tubercle and glioma favor the pons and cerebellum. Syphilis favors the pons, basilar area, and cortex, but is rare in the cerebellar white matter and the centrum ovale. Fibroma and glioma, being interstitial growths, occur in the deep structures. Sarcoma mostly occurs in the ventricles or meninges.

A rapid onset, followed by a stationary period, speaks for tubercle, especially in the first half of life. Glioma and sarcoma are of insidious and steady development. Apoplectic seizures in tumor cases usually mean glioma. Syphilis provokes a rapid onset and course.

Only syphilitic tumors are permanently affected for good by treatment, but it must be said with due emphasis that the iodids are capable of apparently checking sarcomatous growths and frequently cause temporary and misleading benefit in all forms of tumor, probably by favoring the removal of the circumscribing edema.

The fifth question concerns the possibility of *surgical removal.* From the large statistics compiled by Starr, and from later additional data, it can be stated that not more than seven times in a hundred cases of brain-tumor is the growth operable. Unless the neoplasm lies on or in the convexity of the cerebral hemispheres it is not readily approachable. Piollet[1] tabulates forty-eight cases of cerebellar tumor surgically removed. In twenty death promptly ensued; in sixteen improvement with subsequent death from recurrence of the growth; in four almost complete cure, and apparently complete cure in one only. Frazier,[2] from a study of 116 operations of recent date, draws the following data: Recovery, 15 per cent.; improved, 28 per cent.; unimproved, 15 per cent.; mortality, 42 per cent. Cerebellar cysts offer a good operative prospect. The basilar, pontine, and medullary regions are out of the operative field. Cysts and old abscesses are readily drained. Sarcomata can usually be enucleated, tubercles and fibroid tumors can be shelled out, but glioma, from its infiltrating character, and other similar growths can never be entirely removed, and grow again if the attempt is made.

Prognosis.—If ninety-three per cent. of brain-tumors are inoperable and syphiloma is only partially amenable to medicinal treatment, the gravity of the disease is apparent. The great majority of cases run their course within three years, though slow-growing neoplasms may exist almost an indefinite time or may only furnish a post-mortem surprise. The outlook turns upon the nature of the growth and the associated clinical manifestations. The possibility of sudden death should not be overlooked, especially in tumors located in or near the brain-axis.

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[1] "Arch. Provinc. de Chirurg.," 1901.

[2] "N. Y. Med. Jour.," Feb. 18, 1905.

Treatment.—The management of brain-tumors is of two sorts: (1) That directed to the tumor itself, and (2) that to the general physical condition. Removal of the tumor by operation is possible in a small number of cases, and should be done whenever indicated and the conditions are otherwise favorable. This surgical proceeding in the hands of competent men has secured some brilliant results and saved and prolonged life. Oppenheim states that good results have been obtained in about one-half of all well-selected cases. Even in inoperable tumors a wide opening of the skull has relieved pressure, has benefited the mental condition, stopped the headache, and caused the choked disc to subside. It is indicated in at least five-sixths of all cases, according to Knapp.[1] Indeed, in some cases it has seemed to cause the tumor to recede. As above indicated, cerebellar tumors are not the most favorable for operation. The harder and slower growing sorts of brain neoplasms offer the best operative results. Tubercle, sarcoma, and fibroma, or their varieties, can be removed *en masse*, while the limits of infiltrating growths are difficult to distinguish and complete removal is practically impossible. Cysts and abscesses can be evacuated, and if the secreting wall is removed, a practical cure follows. Should the cyst be due to degeneration of a sarcomatous growth, the probability of a return is very great.

In syphilitic growths intensive treatment with mercury and the iodids usually produces prompt improvement. This goes so far that many cases are alleged to be cured, and the shriveled remains of a syphiloma have been found post mortem to testify to the efficacy of treatment and the precision of diagnosis. A large degree of reservation, however, should be maintained in every syphilitic case. As a rule, the cure is not complete. Some residuum of disability can usually be detected, and a constant tendency for the syphilitic process to reappear during the rest of life too frequently keeps these patients fighting the disease as long as they live. In order to secure the best results, both mercury and iodid should be used, either together or alternately, depending upon the urgency of the condition, and both, with careful guarding, should be pushed to the limit of toleration. Explicit directions for the treatment of syphilis will be found in the chapter on Syphilitic Diseases of the Nervous System, Part VI.

As tumors of all varieties have shown at least temporary improvement under the use of antisyphilitics, the practitioner must be guarded in drawing inferences from such therapeutics, and not allow himself to take too favorable a view of the case when this occurs. It is an established rule to use specific treatment in all cases of brain-tumor where there is a shadow of a doubt as to their character. If, after six weeks of vigorous treatment no benefit is obtained, the lesion is pretty surely not syphilitic. Should improvement take place, a continuance of treatment should be persisted in until all symptoms have practically disappeared. A return of former symptoms or further development of tumor indications in the face of specific treatment practically demonstrates the non-syphilitic character of the disease. Horsley contends with much reason that when attackable, even syphilomata should be treated surgically after a fair trial of specific medication, and Coombs Knapp[1] found nine out of ten such operations to have been successful.

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[1] "Boston Med. and Surg. Jour.," Oct., 1899.

The patient's general condition will require constant attention. Tuberculous and other cachectic states have their own requirements. The headache can often be relieved for a time by brisk cathartics and hot baths. Antipyrin and other coal-tar analgesics often control the headache for a time. The opiates are frequently powerless except in extreme doses, and their use should be postponed to the last possible moment. Vomiting yields best to nerve sedatives, such as the bromids, and to measures like hot baths and mustard foot-baths, calculated to decongest the cerebral circulation. In rare cases it is quite unmanageble, even by morphin, and may lead to rapid inanition and death from exhaustion. Convulsions can usually be restrained by the bromids. The continued use of bromids and antipyrin will be found especially valuable in these cases, and a flagging heart may be protected by caffein or strychnin. The optic neuritis can be benefited in syphilitic cases by medicinal treatment. In other sorts of growth it is frequently benefited by operation. Repeated spinal puncture may also yield palliative results. When optic atrophy has once occurred, it is permanent. Finally, by nutritious diet, baths, massage, and general measures the strength of the patient is supported and life prolonged.

CHAPTER XII.

HYDROCEPHALUS.

HYDROCEPHALUS is a term loosely used to designate any undue amount of watery fluid within the skull, and in such a sense is synonymous with dropsy of the brain. Tubercular meningitis, frequently called acute hydrocephalus by older writers, is not the condition in question. Nor are we now to consider the compensatory increase of cerebrospinal fluid occurring in the convolutional shrinkage of old age, or in the cerebral atrophy of dementia, or in porencephalic or anencephalic defects. The question does not pertain to the increase of fluid which marks acute meningitis or tubercular meningitis, or to the edematous state that frequently attends cerebral tumors. Attention has also been called in the proper place to ventricular distention resulting from tumors situated in the posterior cranial fossæ, which mechanically block the venous return through the veins of Galen and the straight sinus. It is desired to restrict the term *hydrocephalus* to a *congenital* or *acquired*, *acute* or *chronic condition* marked by great increase in the amount of cerebrospinal fluid within the skull attended by compression of the brain.

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[1] "Boston Med. and Surg. Jour.," Oct., 1899.

In some cases it is entirely ventricular, constituting *internal hydrocephalus;* in others it is subdural, constituting *external hydrocephalus;* but usually both the subdural and the ventricular spaces are distended.

Etiology.—The causation of hydrocephalus is obscure. Some families seem to be marked by a hereditary tendency to it, as shown by several cases occurring in the same or succeeding generations. Hereditary syphilis has for long been thought to be a competent cause, but in many cases it can be excluded with a reasonable certainty. Its causal relation is supported by Heller [1] and by Titomanlio [2] in careful studies. Alcoholism on the part of the parents is also supposed to have some part in producing the congenital variety, and is doubtless active in some of the late adult cases. Traumatism after birth has produced it, and it has been noted as a sequel of cerebrospinal meningitis.[3] Quincke describes a form of *acute serous meningitis* the symptoms of which do not vary materially from those of the ordinary infectious variety, but which is marked by the rapid production of subdural hydrocephalus and great intracranial pressure.

Morbid Anatomy.—In congenital cases and in those occurring before the cranial bones are firmly united the head is enlarged, sometimes to vast proportions. The *cranial bones* are usually reduced in thickness, often to that of paper, the diploë being absent. At the same time they are frequently much broadened. The sutures are patent, or supernumerary bones are commonly found if synostosis has taken place after the disease has been present for some time. The frontal, occipital, and squamous portions of the temporal bones are displaced outward. The parietals conform to the globular shape of the head.

The amount of *fluid* may be incredibly increased, and as much as three gallons has been noted in a very extreme case of long standing. It is a colorless fluid of low specific gravity, and quite similar to chronic effusions in other fibroserous cavities and closely resembles normal cerebrospinal fluid.

The principal distention is usually in the lateral *ventricles,* which bulge in all directions and stretch out their cerebral walls into a thin lining for the enlarged cranial cavity. The basal ganglia are often compressed and flattened. The ventricular lining is found usually, if not always, in a thickened, granular, hypertrophic condition. It may reach a thickness of half an inch. The choroid plexuses are correspondingly enlarged. This apparent ependymitis often serves to occlude the ventricular aqueducts, so that the third and fourth ventricles may not share in the dilatation or it may be confined to one lateral cavity. In about one-half of the cases connection with the spinal spaces is obliterated. In the congenital and adult cases it is common for all the cerebral ventricles and the cord to be involved. When the third and fourth ventricles are affected, the optic tracts suffer and optic atrophy is common. At the same time the cerebellum, pons, and medulla are defectively developed. The meninges may show little or no change. After *cranial solidification* the anatomical conditions are modified by the resistant character of the skull. The amount of fluid is necessarily less, but the pressure falls more directly upon the cerebrum.

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[1] "Deut. med. Wochens.," June 30, 1892.

[2] Trans. Section on Diseases of Children, International Med. Cong., Rome, 1894.

[3] E. P. Joslin, "Am. Jour. Med. Sci.," Oct., 1900, eight cases.

Symptoms.—Hydrocephalic *enlargement* of the head sometimes renders birth difficult or requires the use of the perforator before extraction can be accomplished. In other and a majority of the cases the condition is insignificant or unnoticed at birth, and appears during the first year of life, especially during the first six months. The head gradually or rapidly enlarges in all its diameters and measurements. An increase in the circumference of the head at a rate of a centimeter daily has been noted, but usually it requires several weeks for that amount of enlargement to take place. The outline of the skull is globular and may overhang the face, ears, and occiput, which are not correspondingly enlarged. The internal pressure is manifest at the bulging fontanels, which are increased in size and connected by wide-open sutures. The *return circulation* of the cerebrum is impeded, and the collateral veins in the scalp and face become distended and strikingly apparent. From the outward tilting of the frontal bones the orbits are directed downward and the ocular globes are often maintained in the same direction, even to such a degree that the cornea is only seen with difficulty. *Optic atrophy* and blindness are frequent. *Nystagmus* and *strabismus* are common. *Fluctuation* is readily obtained on palpation, and the head, in extreme cases, may be translucent.

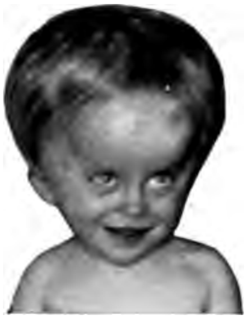

Fig. 100.—Chronic hydrocephalus in a child of four years. Circumference of head, 27 inches.

The hydrocephalic child shows little activity, can not raise its head sometimes, or only does so with the aid of its hands. There is a tendency to peevishness and restlessness or *somnolence* and coma, broken by generalized convulsions and a frequently repeated distressed cry. Older children complain of *pain* in the head. The body and limbs suffer in their nutrition and are unequal to the task of holding up the head and trunk. In some cases *spasticity* develops in the limbs, especially in the lower ones, and some paralytic loss of power is frequently noted. *Vomiting* is frequent and may be provoked by movements or much handling.

If not rapidly fatal by exhaustion, convulsion, coma, or syncope, the disease may come to a standstill or proceed with such slowness that the child is enabled, in some defective measure, to maintain growth and develop its physical and mental faculties. These are both invariably much retarded, so that the body is dwarfish and in great disproportion to the cephalic enlargement. The occasional cases that live to mature years are more or less imbecile, clumsy, and physically defective, though a slight degree of hydrocephalus is not incompatible with mental brilliancy. Many cases of marked dolichocephalia in adults with beetling brow and salient occiput prove the possibility of recovery.

When hydrocephalus is acquired subsequent to cranial synostosis, the symptoms are vague, and only rarely can the condition be deciphered ante mortem. The indications are not unlike those of tumor. It usually follows head injury. Mental impairment, especially of memory, vertigo, vomiting, insomnia, headache, convulsions, and rigidities are encountered. Hemiplegia is rather frequent. The pupils are dilated and stationary. Strabismus is common. Periods of coma are frequent. Death may be sudden or follow coma.

Course.—The disease presents a varied course. The congenital cases may run rapidly to a fatal termination in a few weeks, while others come to a standstill and allow a fair degree of adult development and a comparatively long life. As a rule, marked hydrocephalic cases do not reach maturity, but give out at adolescence and puberty, if not sooner carried off by convulsions or comatose conditions referable to the intracranial pressure. It is extremely rare for them to live beyond thirty. In the acquired adult cases a fatal termination is usual within two or three years, and often sooner.

Diagnosis.—The diagnosis in infantile cases can hardly offer any difficulty if the tendency to cephalic enlargement is noted. The globular shape of the head should distinguish it from the rectangular conformation of rickets with the enlarged and squared forehead and prominent frontal eminences, though patency of fontanels is usually present in both. The appearance of rickety conditions in the long bones and at the costal extremities is also significant. The two conditions, however, may be associated. Before the head shows much or any abnormal increase it is difficult to exclude meningitis, which may, indeed, be the causal condition. The treatment, however, is very similar in both.

In adults the diagnosis is well-nigh impossible, and when suspected can only be confirmed by an autopsy. A condition acquired after complete union of the cranial bones, commencing anywhere from five to fifty years of age,—hyperostosis cranii,—may at first sight mislead. The history will at once differentiate it. Notable hydrocephalic enlargement must begin in very early life. In hyperostosis cranii the size of the head is due to a thickening of the cranial bones, and the bones of the face and spine are usually affected in a similar way. The enlarged head of acromegalia may be distinguished by its late development and the associated deformities of face, hands, and feet.

Prognosis.—The prognosis for life is always grave. The great majority of cases die within a year. A few with enormous heads live for a few years, and in those marked by a stationary condition life is still usually much shortened. The outlook for mentality is also darkened, but must be estimated for the individual case. Mental enfeeblement is the rule, and this may be mere childishness or pronounced imbecility. Epileptoid attacks are of serious import both as to life and mental development.

Treatment.—The treatment of these cases is medical and surgical. Mercurial inunctions to the head and the use of iodine preparations have long been practised. It is probable that some of the good results attributed to these measures are not due to their alleged stimulation of absorption, but to their influence on a syphilitic factor. As a rule, they are of little value, and should only be used when there is suspicion of specific taint. Catharsis and other violent elimination is to be discountenanced, as it only serves to exhaust the patient's diminished strength. When the process is active, the application of cold to the neck and head by ice-bags or flexible coils is valuable. A slight mercurial action, preferably by calomel in minute and frequent doses, should be used. Convulsions require sedatives, especially bromids.

Strapping the head with surgeons' plaster or the application of elastic caps and bandages have been advocated, but are usually intolerably painful and aggravate the pressure conditions. Repeated tappings through the fontanels or by lumbar puncture have been employed with varying results. Occasionally they have seemed to be successful, but lumbar puncture will be fruitless in at least half of the cases owing to the lack of communication between the brain and cord spaces.[1] Only a moderate quantity of fluid should be withdrawn at a time, and the strictest antiseptic precautions must be employed. Some have ventured to withdraw fluid and inject iodine solutions as in the treatment of hydrocele, but the plan can not be advocated. Too frequently the punctures result in a meningitis that carries off the patient. Plans of constant external drainage have been devised, and, while attended in some cases with temporary benefit, the result has been uniformly fatal.

A case has been reported by Rokitansky in which spontaneous rupture under the scalp led to a recovery. The idea has occurred to Dr. L. L. McArthur, of Chicago, to drain the ventricular cavities into the areolar spaces beneath the scalp by the insertion of drainage-tubes or silk into the cranial cavity through a drill-opening above and back of the ear. He attaches the drainage material to the pericranium, and then, securing first-intention healing in his overlying scalp-flap, allows the serous accumulation to flow. A hygromatous swelling forms under the scalp from which absorption seems to be rapid, and the head diminishes in size if the bony sutures are not united. This operation was done for the writer on the case shown on page 267, with immediate and lasting improvement. In two other cases it also demonstrated its utility, but is still on trial and not devoid of danger. Mikulicz and Troje[2] have had favorable results with the same method independently devised. Others have established drainage from the lumbar spinal spaces into the peritoneum. The disadvantage of all surgical procedures is the liability to infection and meningitis and the peculiar tendency of these cases to sudden death from removal of fluid or from the inhibition of the heat-controlling mechanism, which allows the temperature to exhaust the patient in a few hours.

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[1] d'Astros, "Les Hydrocephales," Paris, 1898.

[2] "Centralblatt f. Chir.," Sept. 5, 1896.

PART IV.

DISEASES OF THE SPINAL MENINGES AND SPINAL NERVES.

CHAPTER I.

SPINAL MENINGITIS AND SPINAL MENINGEAL HEMORRHAGE.

SPINAL meningitis is an inflammation of the covering membranes of the spinal cord. The varieties of meningitis ordinarily described have been somewhat arbitrarily based upon their anatomical location. The terms *pachymeningitis* and *leptomeningitis* are respectively employed, as the dura or the softer membranes are principally involved, but a sharp division is clinically impossible, and is not found post mortem. For purposes of description we may consider: (1) *Pachymeningitis*, or external and internal inflammation of the dura; and (2) *leptomeningitis*, or inflammation of the pia. Inflammation of the inner surface of the dura, from contiguity, must involve the leptomeninges more or less, so that the conditions are usually associated, and meningitis, originally external, may finally invade the pia. Owing to the very intimate relation of the pia and the fibrous septa of the cord, association with myelitis is frequent. A mixed form, *meningomyelitis*, is common.

Pachymeningitis Externa Spinalis.—Pachymeningitis externa, or external dural meningitis of the spine is due to chronic irritation and inflammatory conditions invading the spinal canal, and is, therefore, secondary to other morbid states. Vertebral tuberculosis, Pott's disease, abscesses and new growths near the spine, inflammation and purulent collections in the pleuræ, mediastinum, peritoneum, and pelvis, and sacral bedsores may be the source of the meningeal thickening. This gives rise to symptoms mainly by irritation of the sensory and motor nerve-roots passing through the area of disease. When the thickening becomes extreme, as occasionally happens, it may be sufficient to compress the cord and give rise to pressure symptoms and a spastic paraplegia similar to that of a cross-myelitis. The condition is usually due more to the inflammatory invasion of the cord than merely to pressure. There is local tenderness over the spine, shooting or constant pains in the distribution of the irritated nerves, twitching of their muscles, and hyperesthesia in their cutaneous areas, which may go on to anesthesia and muscular palsy if the nerves be sufficiently compressed or inflamed to cause their degeneration.

Anatomically, the dura is found hyperplastically thickened, with much adventitious fibrous tissue, and is frequently covered by a caseous or purulent deposit or involved in a new growth. The various findings, of course, depend upon the nature of the primary disease. When the thickening is extreme, the soft membranes are adherent to the dural tumescence and may be indistinguishable. The cord then shows a constriction, and may, in severe cases of long standing, be very considerably reduced in size at the place of disease presenting local myelitis and secondary ascending and descending degenerations.

The **diagnosis** is usually not difficult if the primary disease is recognized. It may be confounded with myelitis, with which it is often associated late in the case. The clinical history shows a preponderance of pain, spasm, and irritation, a chronic course, and an early absence of paralysis; while in myelitis the rapid onset, the absence of pain aside from the girdling sensation, and the promptly developed paralytic state with early bladder and bowel symptoms are distinctive.

Owing to the serious nature of the causal conditions, the **prognosis** is bad and treatment is practically surgical. The pachymeningitis externa associated with Pott's disease is perhaps the least grave, as the proper orthopedic and surgical management of such cases frequently is followed by practical recovery even when the cord has probably been notably compressed.

Pachymeningitis Interna Spinalis.—Pachymeningitis interna, or internal inflammation of the spinal dura, is described as (1) hypertrophic and (2) hemorrhagic. In reality, these forms are but stages of one and the same process, the thickening and hypertrophy following upon the organization of the hemorrhagic exudate. The term "hematoma of the spinal dura mater" has been sometimes used. The condition is a rare one, and usually the cerebral meninges are also similarly affected. It is most commonly found in general paralysis of the insane and chronic alcoholism.

The portion of affected dura presents on its inner surface a very considerable thickening, which may be a layer of reddish-brown exudate or consist of laminations of fibrous tissue, the apparent result of the organization of successive hemorrhagic exudations. It may attain sufficient size to constrict the cord. The softer, more recent, and reddish or brownish layers consist of fibrin and blood. The distribution is frequently extensive, but in some instances it is confined to a comparatively short vertical extent of the envelope of the spinal cord, and is then more frequently situated in the cervical region. This circumscribed cervical form was first described by Charcot and Joffroy, who give it the name of *pachymeningitis cervicalis hypertrophica*.

Syphilis, trauma, alcoholism, and exposure are regarded as competent causes, and hence it occurs, as a rule, in adult males, though some cases in children are recorded.

The condition is essentially chronic and of slow onset. At first irritation of nerve-roots gives rise to local pain and hyperesthesia over the spine and in the peripheral distribution of the spinal nerves of corresponding origin. This is followed, months or years later, by gradual loss of power, atrophy, and anesthesia in the corresponding parts, and as compression upon the cord is produced spastic symptoms appear below, with increased reflexes, rigidity, and paraplegia, leading sometimes to exhaustion and death. Some cases present stationary periods, and a few recoveries are claimed. The muscles of the forearm are not uniformly affected, the flexors being most impaired. This results in a peculiar deformity that is striking and almost characteristic. The small muscles of the hand usually suffer and both arms are commonly affected, though not usually in equal degree.

The *diagnosis* is difficult when the dural involvement is of general distribution and cerebral symptoms are present, as the spinal features are overshadowed. Diseases of the spine, progressive muscular atrophy, cross-myelitis, tumor, and external pachymeningitis must be excluded. An operation may be required to differentiate the external dural inflammation. It presents, except in syphilitic cases, the best chance of favorably influencing the condition and preventing destruction of the cord. In the desperate situation that is presented and with the courage given by asepsis, it may the more reasonably be resorted to early. Where syphilis is suspected, specific treatment should be persistently tried.

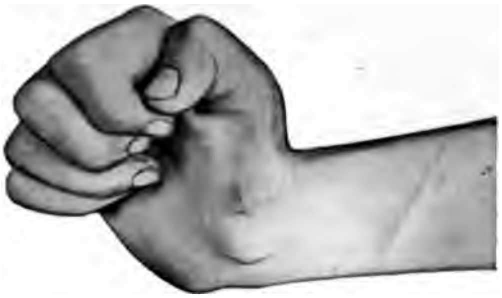

Fig. 101.—Position of hand in pachymeningitis cervicalis hypertrophica.

Acute Spinal Leptomeningitis.—Acute spinal leptomeningitis, or inflammation of the pia mater, is due to infection. It usually involves the inner surface of the dura, and commonly extends to the peripheral substance of the cord.

Etiology.—The infection of cerebrospinal meningitis in epidemics of the disease falls sometimes only on the cord, and the infective nature of the attack is obvious. In those cases, however, that are attributed to exposure, insolation, rheumatism, and other occult conditions, the infection is less readily comprehended, but in all probability is equally in operation, being favored by the physical conditions mentioned. In some recent cases the discovery of the pneumococcus and the meningococcus proves the identity of the infection with that of the cerebrospinal type, to which the reader is referred. The association of cases with septicemia, pyemia, and other infectious blood-states points again to infection, and in the lymph and spinal fluid of these cases abundant pathogenic organisms have been observed. In some instances the spinal disease is an extension from the cerebral meninges, the cervical portion of the cord being usually the only part involved, but the entire dural sheath may be filled with pus from within the cranium. Traumatism of the membranes by vertebral dislocations, strains, and severe concussions may incite a leptomeningitis over a limited area, from which it may extend, or in which a virulent infection may find a suitable field for development. Surgical operations upon the spine, penetrating wounds, sacral bedsores, and communication with adjoining suppurative foci may furnish the infection. Tuberculosis is a common cause, but the resulting meningitis is rather less acute. This is the case to a greater degree in syphilitic inflammation, which has a marked tendency also to remain localized.

Morbid Anatomy.—The disease is usually of wide extent, the infection traveling rapidly through the arachnoid spaces, and finding in the spinal fluid an excellent medium for its propagation and extension. Congestion of the pia, of the adjoining inner surfaces of the dura, and of the cord, marked by increased vascularization and an increase of spinal fluid, passes into inflammation, with dulness of the membranes, opacity, thickening, and an exudate, varying in color from opalescent to puriform, and of corresponding consistency. The microscope shows the diapedic elements of inflammation and often numerous bacteria, including at times the pneumococcus of Friedländer. Tubercles here correspond to their histological and bacterial characters on other serous surfaces. For a time the somewhat resistant pial covering of the cord and nerve-roots protects these structures, and especially is this true in the purulent form of the disease. Usually the periphery of the cord and the roots show the inflammatory invasion, with corresponding changes in the nerve-fibrils, neuroglial framework, and vessels. In cases reaching a convalescent or chronic stage, adhesions form between the cord and the dura, obliterating the arachnoid space over more or less extensive areas, distorting the nerve-roots, and sometimes changing the outlines of the cord itself. If cord-softening has taken place as a result of the meningomyelitis, degenerations of its conduction tracts and localized destruction of its gray matter are found. Large quantities of spinal fluid usually mark these late cases, causing, with the irregular adhesions, a sacculated condition of the dura.

Symptoms.—The abrupt *onset* of the disease may be preceded by a day or two of malaise and slight anorexia, but sometimes no invasion period is present. A sharp chill is followed or attended by great *pain* in the back and darting pains around the body or down the limbs. In children vomiting is a common symptom, and *convulsions* may be present. *Tenderness* is at once developed over the spine. It is easily detected, when not prominent, by the use of a sponge dipped in hot water or by percussion. Spasm and *rigidity* of the muscles appear, causing stiffness of the neck and back, sometimes notable retraction of the head and vigorous opisthotonos. Fixation of the limbs upon the body is more or less marked, with a tendency to flexed attitudes. Retraction of the belly results from implication of the abdominal muscles. Sometimes difficulty of breathing is occasioned by involvement of the chest-muscles. Dyspnea, Cheyne-Stokes respiration, and cardiac symptoms follow medullary implication. The cramps in the muscles are painful, and yet tenderness and hyperesthesia in the limbs prevent manipulations and passive movements. The rectum and bladder are the seat of similar spasms, which may cause constipation and retention of urine, with frequent annoying and ineffectual expulsive contractions of these viscera.

Pulse and *temperature* are fickle, sometimes being subnormal, sometimes increased, and more often divergent; for instance, a subnormal temperature with an accelerated pulse. The lack of uniformity in their range is especially valuable in diagnosis, even when the cerebrum is apparently not involved. A temperature of 103° F. is not uncommon. *Vasomotor* paralysis is usually shown by the vivid, persistent, but slowly developed line which follows every stroke of the finger-nail upon the skin, and from the same cause the limbs may be congested and even slightly edematous. At first, for a day or two, *reflexes* are inclined to be increased and later may be wanting.

Cases which outlast the acute symptoms develop paralysis, anesthesia, atrophy, and contractures in proportion as the cord and nerve-roots are affected. Paraplegia may result, presenting the features of a cross-myelitis, with bladder-paresis, bedsores, increased reflexes, and spasticity. Symptoms vary with the location of the disease, but its tendency to involve the entire spinal apparatus is marked, and indications of its effect upon all spinal segments are to some degree present in a majority of instances. Some regions situated in the focus of the inflammatory action show early and emphatic involvement; those at a distance may be disturbed very little. Yet in some purulent cases, where the dural sheath is greatly distended through its entire length with the large accumulation, the pia protects the cord and nerve-roots from infection, so that pressure symptoms alone may be present.

Course.—Some cases terminate fatally within a day or two; others last a fortnight, and may then end fatally or recover. The nature and virulence of the infection are determining factors, as is the location of the disease. Extension upward or early involvement of the high levels of the cord tend to an early fatal issue. Complete recovery is rare and the conditions resulting from secondary myelitis are of long duration and usually last the life-time. The tubercular and syphilitic varieties, as already indicated, run their course less rapidly, and the latter is capable of material modification by treatment. Even rare tubercular cases may get well.

Diagnosis.—The diagnosis depends upon the rapid onset, the pain in the back, the radiating pains, the rigidity, the increase of pain on voluntary movement, the hyperesthesia, and the fickle temperature and pulse. From myelitis it is distinguished by the paralysis and lack of pain which characterize the cord-lesion, but the frequent association of the two is to be kept in mind. Hemorrhage into the subdural space, owing to the irritation of the nerve-roots, presents very similar symptoms, but is extremely rapid in onset, usually following traumatism or a strain, and develops meningitis in a short time thereafter. Hemorrhage into the spinal cord gives instantaneous symptoms, immediate paralysis, and may be practically devoid of pain. The rigid form of tetany may present a very close counterfeit, but its long duration, remissions, and amenability to spinal sedatives, the absence of spinal tenderness and shooting pains, the possible history of previous attacks, and the usual irritability from pressure upon the nerve-trunks and arteries should differentiate it. Tetanus may be mistaken for spinal meningitis. The early trismus, the excessive hyperesthesia, the fever of onset, the paroxysms of spasm, and the frequent history of traumatism point the way to diagnosis. Muscular rheumatism and strain present a very superficial resemblance.

Prognosis.—The outlook as to life is always serious and is grave in proportion to the acuteness of the onset, to the virulence of the infection, to the implication of the upper portion of the cord, and to the height of temperature. The estimate is also to be guided by the previous condition of health and the age of the patient, children and the aged quickly yielding to the disease. Traumatic and surgical infection is less serious than auto-infection. The possibility of the removal of sources of infection is of some importance as to ultimate results, provided the patient survives the acute stage. The late results, due, for the most part, to permanent changes in the cord, are usually beyond the hope of marked improvement.

Treatment.—Complete and absolute quiet is to be insisted upon. The patient should be kept upon the side or face, if it is possible to do so without increasing the cramps. The partial knee-elbow posture over a mound of firm pillows will often be found very comfortable, and at the same time will afford the best opportunity for local applications. These, at first, should be strongly counterirritant, as the thermocautery, blisters, or detergents like leeches, vigorous dry cupping, or wet cups in robust or plethoric individuals. Should myelitis be associated, less active measures are indicated, and the skin must not be broken or highly irritated, owing to the tendency to bedsores. A hot bath and pack at the onset with active catharsis have seemed to do good. Sedatives, especially spinal sedatives, are frequently required to control the spasms and anodynes to relieve the pains. A thorough course of mercurial inunctions over the spine has strong advocates, the quantity used being sufficient to produce slight ptyalism. Owing to the reflex irritability, these rubbings must often be impossible, and the therapeutic value of mercury in the acute stage of non-luetic cases is open to question. Quincke's lumbar puncture has here the same indications as in the cerebrospinal form. Iodid of potassium and ergot are also at this time of little or no value. The ice-bag to the spine is one of the most serviceable measures, but is rarely tolerated long by the patient, and its intermittent application is useless. It should always be tried. As the active stage subsides, light cauterizations with the Paquelin apparatus, mild sinapisms applied for six or eight hours, and the hot-spray douche seem to assist the reparative efforts of nature. Cerebral symptoms usually mean the implication of the brain-coverings, the spinal features become of secondary importance, and the treatment is that of cerebrospinal meningitis. The paralysis, contractures, and other late results of the myelitis are to be managed in accordance with the rules of practice in that disease.

Chronic Spinal Leptomeningitis.—The chronic form of inflammation of the soft spinal membranes is usually the sequential stage of an acute attack, but may follow alcoholism, syphilis, or tuberculosis. Its existence as a primary affection is open to some doubt, but a very slowly developed leptomeningitis may follow concussion, though it is impossible in such a case to exclude immediate slight histological injuries of which the later inflammation is a natural development. The formerly much used term "chronic meningitis," which was applied to every group of obscure subjective symptoms, however remotely referable to the spine, needs no mention.

The symptoms are practically those of the acute form much reduced in intensity, and are dependent upon similar causes. Pain in the back predominates, and spasm is insignificant or absent. The radiating neuralgic pains are especially pronounced, and paresthesiæ are prominent. Their distribution depends upon the nerve-roots involved and the location of the inflammation, which is much more circumscribed than in the acute form. The late manifestations are those due to neuritis originating in the roots, and myelitic symptoms are comparatively infrequent.

The anatomy of the disease is very little known, as opportunity for post-mortem examination rarely occurs, but more or less extensive fibrous thickening, or adhesions between the pia and dura which constrict the nerve-roots, may be found, and may girdle the cord. Degeneration of the spinal nerves traversing the lesion is not rare, and this accounts for the herpetic and other cutaneous symptoms which are occasionally noted.

The prognosis will be guided mainly by the effect of treatment, but a complete recovery is very rare. Each case must be carefully estimated by itself.

The treatment in syphilitic cases consists in the heroic management of that disease, and iodids and mercury in small doses are also the most efficient drugs in non-luetic cases. General measures are of service, and persistent counterirritation over the spine, preferably by Paquelin's cautery, is the most valuable local measure. Sometimes rest in bed and the ice-bag to the spine are of distinct value. Sedatives and analgesics are often required.

Spinal Meningeal Hemorrhage.—Spinal meningeal hemorrhage is either *extradural*, in the vertebral canal, or *subdural*, within the dural sheath. It is frequently associated with intracranial hemorrhage and with hemorrhage into the substance of the cord, but also occurs independently.

Etiology.—Meningeal hemorrhage occurs frequently at birth in protracted and difficult labors, and is then almost invariably associated with extensive hemorrhage within the skull. It has been considered under the cerebral palsies of childhood. Spontaneous hemorrhage is very rare, but occurs in adult life at all ages. Disease of the meningeal vessels is sometimes the immediate cause, but in the great majority of cases it is induced by traumatism. It may be caused by direct blows or falls upon the back, shock communicated through the lower limbs, vertebral fractures and dislocations, penetrating wounds and even by severe muscular spasm, as in tetanus, convulsions, and violent chorea. No doubt syphilis, arteriosclerosis, purpura, scurvy, and other hemorrhagic states favor it. The blood sometimes comes from a thoracic aneurysm which has eroded the vertebræ and ruptured into the spinal canal or dura, or from one situated on the vertebral or basilar arteries. Hemorrhage into the cerebral meninges may find its way below the foramen magnum, and in the same way a spinal hemorrhage may invade the cranium.

Morbid Anatomy.—In *extradural* cases the clot usually originates from the rich plexuses of veins that line the vertebral canal. It may be of considerable size and extend through the intervertebral foramina. The most common location is in the cervical region. The dura is stained and infiltrated, and the cord may exceptionally be compressed. Effusions of blood *within the dura* vary much in size. The blood usually comes from the pial vessels, and consequently, as a rule, involves the cord. Complete flooding of the dural sheath is almost always due to intracranial hemorrhage or rupture of an aneurysm. A small hemorrhage tends to remain localized and to surround the cord at the original point. It discolors and compresses the cord and after a few days produces inflammatory changes in the meninges. In the same way an annular myelitis may be induced.

Symptoms.—The symptoms are practically the same in both extra- and subdural hemorrhage. The onset is ordinarily abrupt and the early symptoms depend upon irritation of the meninges and nerve-roots. There is great pain in the back, which often radiates along the implicated nerves, girdling the body or running down the extremities. Tingling and formication are complained of, and paralytic symptoms below the level of the lesion, loss of power, and diminished sensation are induced. Bladder and bowel symptoms shortly appear. There is ordinarily some spinal rigidity, which may develop into opisthotonos, and convulsions are not infrequent. Symptoms are gradually developed unless the hemorrhage is due to severe traumatism or to flooding of the vertebral canal by the rupture of an aneurysm. In crushing injuries, spinal fractures, and dislocations the cord is almost invariably injured, and hemorrhage, if present, adds very little to the symptoms. From the onset to the full development of the paralytic features from one or two to forty-eight hours, or even more, may be required. The symptoms, therefore, greatly resemble those of spinal meningitis, which usually is added after a few days, and its invasion is often marked by a distinct aggravation of all the symptoms.

Cerebral symptoms are only present when the cranial contents are simultaneously affected. Death is likely to occur early when the symptoms have reached their height, or during the secondary meningitis.

Diagnosis.—In cases of insidious onset without definite symptoms, the diagnosis at best can be but conjectural. When hemorrhage follows traumatism, the distinguishing trait is a gradual development of the symptoms within a few hours. Injuries that affect the cord-substance produce instantaneous loss of function, but a meningeal hemorrhage may be, and often is, associated with hemorrhage into the cord. Jacobi has also obtained blood by spinal puncture in two cases of injury of the spine.[1] From meningitis the chief distinction is the much more rapid development in hemorrhage and the history of a competent cause. The *localizing diagnosis* is taken up in subsequent chapters, to which the reader is now referred.

Prognosis.—The outlook is always most serious. As the paralytic features develop, there is a likelihood of death from interference with respiration by paralysis of the chest-muscles. The intense pain and suffering also serve to exhaust the patient. The first danger being passed, secondary inflammation is likely to terminate the case fatally. Hemorrhage in the cervical region is, of course, more ominous than when situated lower down. If the patient survives the first fortnight, improvement may be confidently expected, and this may practically be complete, though some disability remains, as a rule, and it may be of an extreme degree.

Treatment.—At first the most complete rest on the face or side with the spine elevated should be secured. An ice-bag to the back is a valuable measure if persistently and thoroughly used. Venesection to lower the blood-pressure has been used, but will not find many brave enough to employ it. Local wet cups with free flow of blood have also been employed, but are of doubtful value. Remedies that increase the coagulability of the blood may be exhibited, but ordinarily the flow of blood is of but a few moments' duration, and no time is given for their action. If the diagnosis is fairly certain, the spinal canal should be aseptically opened and the dural sheath also incised. The operation as now done adds nothing to the gravity of the case, and has enabled the surgeon to remove clots with the best results. The secondary meningitis and the sequential palsies are to be treated on their own indications.

CHAPTER II.

INJURIES AND DISEASES OF SPINAL NERVES.

THE spinal nerves, unlike the cranial group, are both motor and sensory. In addition they contain the vasomotor supply, and through them is exerted the trophic influence of the spinal centers over the peripheral apparatus. Their injury or disease is, therefore, marked by perversion or abolition of these functions, and gives rise to groups of symptoms anatomically coextensive with the distribution of the particular nerve or nerves involved. We should bear in mind that the fibers making up a nerve-trunk are cellular elements,—prolongations from cell-bodies, of which they form an integral and functionally essential part. When we injure a motor fiber in a nerve-trunk, we injure a motor cell. In other words, we injure the lower motor neuron. We will first consider nerve injuries and diseases in a general way, and then the particular conditions which pertain to such states in special nerves.

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[1] "Amer. Jour. Med. Sciences," Oct., 1900.

Division of Nerves.—Spinal nerves are frequently divided by incised and bullet wounds, sometimes by crushing accidents, by simple and compound fractures, and rarely by dislocations. Causes acting more slowly may end in the destruction of a nerve, but a neuritis or degeneration is usually, if not always, first induced. After a nerve is divided the peripheral portion degenerates, and the process is called *secondary degeneration.*

The immediate *symptoms* are loss of motion, sensation, and muscular reflexes in the distribution of the nerve. Shortly afterward, within forty-eight hours, the muscles supplied by the injured nerve lose their tonicity and then progressively waste. Vasomotor paralysis appears and trophic disturbances in the cutaneous area of distribution are marked by a thin, shiny skin, with atrophic hairs, nails, and other epithelial structures. There is also a lowered vital resistance to infection, and healing processes are slow and faulty. Even the joints are sometimes affected, and bony growth in the young is retarded. Electrical stimulation through the nerve fails completely. The muscles lose their responsiveness to faradism, and the increased galvanic irritability which at first appears gradually subsides and is finally lost. The electrical changes constituting the *reaction of degeneration* are more fully described in Part I, page 45. In the extremities the unapposed antagonist muscles then draw the joints into fixed, rigid positions. Muscular contractures develop and still further tend to deform the part.

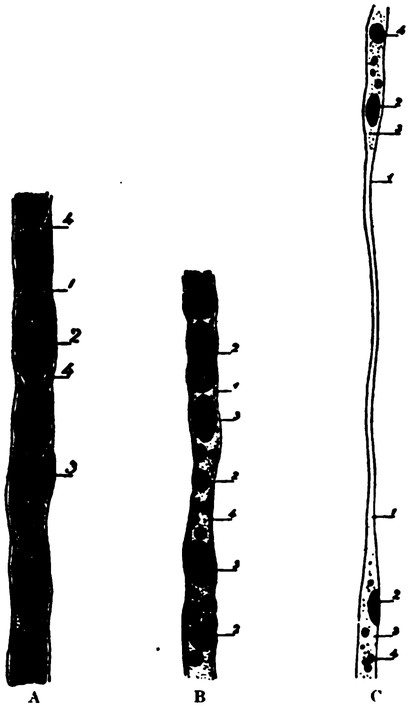

Fig. 102.—Nerve tubules in peripheral portion of a divided nerve—A, two days after section; B, four days after section; C, eight days after section. *1*, Sheath of Schwann; *2*, myelin; *3*, nuclei; *4*, protoplasm (Ranvier).

The *histological changes* that take place in the *distal portions* of the divided nerve are as follows: The nuclei of the internodal nerve-cells swell, and their protoplasm becomes increased in quantity, but changed in character, as it no longer stains so actively as in health. The nuclei also become segmented, and with the increase in protoplasm encroach upon the myelin and displace it. The nerve-fiber then shows an irregular beading of the myelin, and at the points of greatest constriction the myelin finally separates transversely, and the axis-cylinder is divided at the same time and in the same way. This process takes place uniformly throughout the length of the divided nerve below the lesion. The segmented myelin becomes more and more aqueous, escapes in part through its sheath, and is absorbed. The nuclei cease proliferation, and the nerve-fiber is left a mere connective-tissue filament, except at irregular intervals, where remaining globules of myelin may distend it.

The segmentation of the myelin in man reaches the point of complete division and consequent rupture of the axis-cylinder at about the end of the third day after nerve-division. At this time electrical conduction or excitability in the nerve is also lost and muscular tonus is destroyed. The entire process of degeneration after nerve-division may be completed within three weeks.

Above the point of division *the central stump* degenerates for about a third of an inch only, but there is reason to believe that the cell-body is also disturbed (Marinesco).

The *muscles* supplied by a divided motor nerve are deprived of the trophic influence of the spinal center as soon as division takes place, and the axis-cylinder below the lesion is equally deprived of the trophic support of the cell-body. The sarcode elements waste. The cross striations become less well marked and are closer together; the muscle-fibers become narrowed, cloudy, granular, and sometimes fatty. There is proliferation of the connective-tissue elements of the muscles, which further strangulates the muscle-cells, and eventually a condition of fibrosis or cirrhosis is developed. This has a natural tendency to contract and shorten, and explains the fibrous, tense, cord-like structures and deformities found in such cases of long standing.

The electrical changes that occur in the muscle are also described in Part I, page 46. Faradic muscular contractions are lost within a few days, but at first, for about a week, the muscles react to galvanism even more freely than in health; then the galvanic excitability of the muscle is diminished and disappears.

When the ends of a divided nerve are brought together under proper conditions for healing, *regeneration* may occur in the peripheral portion even when degeneration is complete and of long standing. Bowlby has noted one case of regeneration after fourteen years' division. In nerves freshly divided and at once united, the functions of the nerve are restored in a few days or weeks. After degeneration has been established regeneration is a slow process, requiring from two months to several years, depending upon the condition of the peripheral segment and the surgical features of the given case. According to Tizzoni, Kennedy, Bethe, Ballance, and Stewart,[1] regeneration in a divided nerve is dependent upon proliferation of the neurilemma cells. At the end of three weeks spider-like neuroblasts are seen sending out beaded axis-cylinder processes in both directions from opposite poles, which at the end of the fourth week may overlap and anastomose. A new medullary sheath appears about the eleventh week. Neither axis-cylinder nor medullary sheath attains full maturity unless the distal segment is joined to the proximal nerve-stump. Nonmyelinated nerves, as, for instance, cerebral conduction tracts, do not regenerate, lacking the necessary cellular elements required for that process. The muscles in turn regenerate, and slight volitional motion returns before electrical responses can be obtained with the usual tests. These appear, as a rule, shortly after voluntary power is manifested, and while it is still very slight. Sensory conduction is usually restored before muscular action. The muscle reflexes are the last to reappear.

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[1] Macmillan & Co., 1901.

It need scarcely be added that the only *treatment* for a divided nerve is surgical suture. Whenever the distal extremity can be aseptically united to the central end, and in proportion as it is done promptly after the division, the prospects for a return of function are good. In cases marked by degeneration, electrical stimulation should be early and persistently employed, even if no motor response is obtained. In cases of long standing and in instances where it is impossible to effect nerve suture, the implantation of the distal segment into a neighboring nerve of similar function is frequently followed by a return of functional and volitional control.

Neuritis.—Peripheral nerves are subject to inflammatory action of every grade of severity, arising from an extraordinary number of causes. Many toxic conditions and cachexiæ cause widely distributed and usually bilateral symptoms, due to degenerative states in the peripheral nervous apparatus, producing a so-called *multiple neuritis*, the consideration of which is reserved for a separate chapter. We have here to deal with a nerve inflammation or degeneration arising from local causes. The particular cause, however, may be the topical feature of a systemic disease, as when a nerve is involved in a syphilitic tumor or a gouty, nodular thickening. Consequently the lesion in question is confined to one nerve or to several that are anatomically related.

Etiology.—Slight *compression* or *contusion* may cause temporary or persisting disturbance in the function of nerve-trunks,—a fact made familiar by knocking or pressing upon the ulnar at the elbow or by pressing on the sciatic in sitting. If the compression or contusion is of sufficient severity, the nerve may be an indefinite time in recovering, and long-continued pressure or extreme contusion may, like actual division, induce a complete degeneration. Dislocations, fractures, the formation of callus, violent muscular contractions, strains, cramped positions or continued pressure in sleep, coma, surgical anesthesia and extreme prostration, pressure from the continued use of surgical appliances, crutches, trusses, and constricting shoes or garments are among such causes. A nerve may be *wounded* or *infected* in surgical and accidental ways, and by the hypodermatic needle. It may be *invaded by extension* from a neighboring inflammation, as in arthritis, pleurisy, meningitis, bedsores, abscesses, or any other inflammatory focus. Exposure to *cold* is especially active, as in the facial nerve and others that are superficial or contained in rigid canals and passages. New *growths*, like cancer and gumma, and infiltrations in leukocythemia, cancer, leprosy, and syphilis may cause a neuritis.

There are also a number of *predisposing causes*, some of which are active in the etiology of multiple neuritis. Here may be enumerated gout, rheumatism, diabetes, exposure to cold, alcohol, lead, and cachexiæ generally. In such conditions an exciting cause acts more readily and repair takes place more tardily.

Morbid Anatomy.—The anatomical changes in a neuritis vary according to the intensity of the process and the particular elements in the nerve-trunk that are most affected. When the nerve-fibers are mainly involved, we speak of a *parenchymatous neuritis*. This form is rarely secondary to a local injury, but sometimes so arises. It is the common form in the multiple neuritis of systemic states. When the fibrous structure of the nerve-trunk is most affected, the neuritis is called *adventitious*. In the case of inflammatory extension from adjoining structures the sheath or surface of the nerve is usually first involved, and the condition is then distinguished as a *perineuritis*.

An acutely inflamed nerve looks reddened and presents increased vascularity. There is sometimes distinct edema, or a jelly-like infiltration of the sheath and adventitia may be seen. This is ordinarily confined to a limited extent of the nerve, or may be seen at numerous points. It is particularly likely to be present where the nerve is superficially placed over firm structures, as where a nerve passes over bony prominences or is tightly enveloped in fascial or bony tissues. From pressure or irritation thus arising, changes of corresponding degree take place in the nerve-fibrils. When the neuritis or even the contusion is sufficiently severe to disrupt the myelin for a few hours, we have a descending degeneration below the lesion, which then presents the morbid appearances described in nerves after complete division. In the case of neuritis, however, it is common for some fibers to escape, and to appear practically normal in the cross-section of the degenerated nerve.

As a rule, the inflammatory and degenerative process is confined to the injured portion of the nerve and the parts beyond, but occasionally, and then usually in the case of infected wounds, the neuritis extends upward, and if it reaches the plexus of origin may there involve other nerve-trunks related to it. This ascending form is sometimes called *neuritis migrans*. In cases of long standing the adventitia is increased and a fibrous thickening of the nerve may result, which persists even after restitution of function has taken place. In unrecovered cases the degenerated nerve may be reduced to a mere fibrous filament.

Infiltration in syphilis and cancer is identical with these processes elsewhere, and may cause distinct tumors in or on the nerves. In leukocythemia and leprosy there is merely an infiltration, which in the latter may contain the characteristic bacilli. Perhaps the same may be true in neuritis arising from tubercular ulcerative lesions.

Symptoms.—The symptoms of neuritis vary greatly. They may be mainly those of *irritation* or those of *destruction* of the conducting functions of the nerve-fibrils. Ordinarily both are present. Local conditions at the point of original nerve injury, such as swelling, contusion, or laceration, may be added. The *onset* is ordinarily abrupt, in the case of traumatism instantaneous. When the nerve is insidiously invaded the symptoms develop with corresponding slowness. The *sensory disturbances* are usually the most prominent at first. All varieties of paresthesia are complained of: Formication, numbness, tingling, burning, heaviness, deadness, coldness, etc. There is considerable pain in severe cases at the site of inflammation, and in the muscles and skin to which the nerve is distributed. The nerve-trunk and the supplied muscles are usually tender on pressure. The nerve tenderness is particularly prominent where the trunk is rigidly held by fibrous tissues or passes over bones. The nerve may sometimes be palpably enlarged. Pressure upon it by muscular action may also elicit tenderness and increase the pain. The cutaneous area of its distribution presents hyperesthesia or diminished sensitiveness, or both. Often there is a feeling of painful sensitiveness when tactile perception is actually blunted. After degeneration starts in the nerve, anesthesia is present in the skin supplied by the nerve, though irritation at the site of original lesion may still lead to complaints of peripheral pain, constituting *anesthesia dolorosa*. The striking feature of all these sensory disturbances is their constancy and uniformity in a given case. Stabbing pains and momentary paresthesia are wanting. As the inflammation progresses the conductivity of the nerve decreases, hyperesthesia becomes anesthesia, and paresis passes into paralysis. The anatomical distribution of the sensory features is very important. Often it can be mapped out with the greatest precision, but at first usually the margin of the area of disturbed sensation is not so sharply defined and the entire limb may be painful and sensitive.

The *paralytic muscles* are those which are supplied by the given nerve. Opposing muscles may lack force from the loss of proper balance, or their use may be inhibited by pain. Delicate finger-motions are rendered clumsy by the sensory disturbance or the loss of power, or by both combined.

If the lesion be sufficiently severe to set up degenerative changes in the nerve, we find the *trophic losses* in the skin and muscles described under Division of Nerves, page 279. These may be, and often are, present in a modified degree when motor and sensory functions are not completely gone. In all cases of long standing they are sure to appear. With them go the various other symptoms already described: the *loss of muscle reflexes*, the *electrical changes* of irritation, degeneration or destruction, and the development of contractures. In complete division of nerves the *nutritional disturbance* is usually a simple atrophy, but in neuritis there is a wider range of dystrophic conditions. These are most marked in the hands and feet and most apparent in the dermal structures, though the muscular wasting is sufficiently apparent and often most striking. In some cases there is much obscuring edema, due to the lack of vascular and muscular tone. In these cases and in those in which redness and profuse perspiration are present, the dermal epithelium and the hairs and nails often take on an excessive growth. As a rule, the nails become roughened, scaly, and strongly curved. The cushions at the tips of the fingers waste and the nails may curve over toward the palmar surface, like claws. About the base of the nails the skin is often thin, glazed, and red, and the entire digit may be reduced to a tapering, stick-like appendage, closely covered with atrophic skin, which no longer shows the natural folds and wrinkles about the articulations. Abrasions do not heal readily. Herpetic eruptions are likely to appear, and deep ulceration may follow unless unusual care be exercised to prevent irritation and infection. In some cases the nails are cast off.

From inaction and the tendency to contracture in atrophied muscles and their unapposed antagonists, the range of joint-motion in chronic cases is usually diminished. Trophic disturbances in the joints are also encountered, with the formation of adhesions, and false ankylosis. In rare cases joint-effusion and other neurotic *arthropathies* have been seen.

Should the neuritis ascend to the plexus and implicate other nerves, similar conditions are induced in their peripheral parts.

Diagnosis.—The diagnosis of a neuritis presents usually but little trouble. If the dysesthesia be confined to the anatomical limits of a given nerve or group of nerves, and the muscular disability is limited to the muscles innervated by the same nerve, the conclusion is obvious. Too much importance can not be given to the study of the cutaneous distribution of the sensory disturbance. If this area corresponds with that of any special nerve (see Figs. 15 and 16), suspicion of a neuritis or nerve injury should be at once aroused. If the corresponding reflexes are gone, or in slight and rare instances are even exaggerated, and if the degenerative or irritative electrical responses are present, the suspicion is confirmed. Sensitiveness of the paralytic muscles and of the supplying nerve-trunk are also important indications, and occasionally the thickened nerve can be felt. A wound or contusion over the nerve is of capital significance. The question often arises in traumatic cases as to whether the nerve has been completely divided. If the reaction of degeneration is present three or four weeks after the injury, the nerve is not totally divided. In the case of division no electrical responses are obtainable after a month. Many cases of neuralgia are confounded with neuritis. The distinction depends mainly on the darting, fleeting, changing character of neuralgic pains and the presence of the painful points of Valleix. Muscular wasting or electrical changes and altered reflexes demonstrate a neuritis.

Prognosis.—The course of a neuritis depends upon its cause and the amount of damage done the axis-cylinders of the nerve. The general tendency is to restoration of healthy function as soon as the cause is removed. A slight neuritis may pass away within two or three weeks, but if the lesion has resulted in degeneration of the nerve, months will be required for the regenerative process. The faradic muscle responses afford a good basis for estimating the probable duration of a case. If the muscles contract fairly to a moderate current two weeks after an acute attack, the disease will probably not last over a month or two. If they do not respond to a strong faradic shock, six to nine months will be required. In long-standing cases the persistence or reappearance of faradic excitability is a favorable sign for complete recovery. On an average, traumatic cases do better than those arising from extending inflammations, especially if they are of a septic character. Should there appear an upward extension of the neuritis the outlook is less favorable. The presence of any predisposing cause also diminishes the prospect of early recovery, and when the neuritis is due to a new growth the prognosis is unfavorable except in syphilitic cases.

Treatment is naturally first directed to any discoverable cause, and the predisposing conditions must not be neglected. Gout, rheumatism, lead infection, nephritis, diabetes, tuberculosis, the use of alcohol, the presence of anemia, malaria, or any cachexia must be corrected as far as possible, and the general physical health brought to its best level. Wounds, septic inflammations, and new growths require surgical intervention. Pressure by crutches, clothing, and surgical appliances must be discontinued. Pain is ordinarily the most pressing complaint. It must be controlled as far as possible by securing complete rest, in severe cases by immobilizing the parts. Muscular action, rude massage, and vigorous electrical stimulation produce pain, work harm, and should be avoided. On the other hand, gentle passive movements and muscle kneadings that do not cause discomfort, repeated daily or twice daily, are of distinct service in maintaining the nutrition of the parts that are temporarily deprived of their proper innervation. To this end the employment of electricity should be instituted, if possible from the first day. Ordinarily the galvanic current should be used, as it is least painful and most active. Only slight muscular contractions should be produced, and fatiguing the muscles must be avoided. If the faradic coil can be used without causing pain and the muscles respond to it, there is no objection to its use. The purpose to be kept in view is to maintain the responsiveness of the muscles to their weakened innervation, or, if that is cut off, to maintain them in the best possible nutritional and responsive state for the return of the slightest influence of the spinal cord that is able to first reach them through the blocked conduction of the injured nerve. These local measures also serve to overcome, so far as may be, the vasomotor and trophic disturbances in the skin.

The use of anodynes calls for mature judgment. They should be postponed to the latest moment, as in cases demanding them a protracted attack is commonly in hand and the danger of installing a drug habit is very great. Counterirritation by sinapism, thermocautery, or small blisters over the nerve is useful in the early stages if it can be made reasonably near the point of inflammation. It should never be made in the cutaneous areas supplied by the affected nerve, as it is likely to produce uncontrollable ulceration.

In *chronic cases* massage, douching with hot and cold water, electricity, and counterirritation are of great value. Strychnin in large doses and hypodermatically sometimes is of use. The use of iodids or of mercury is of questionable utility unless indicated by constitutional conditions, though mercurial inunctions along the course of the nerve have seemed to assist in some refractory cases. Contractures and distorted joints should be treated orthopedically. Muscles that are wasted and weakened may be of some service if not placed at complete mechanical disadvantage by flexed joints and overacting, strong antagonists. Dropped foot, dropped wrist, flexed knee, and flexed hip should be obviated by early and persistent attention, by massage, and, if need be, by splints and fixation apparatus. Secondary contracture sometimes appears in the muscles of the extremities after regeneration of nerves injured by neuritis, just as it does in the face, but the balance is less delicate and distortion does not result so readily.

Nerve-tumors.—Spinal and cranial nerves may present new growths of various sorts. In one group may be included those neoplasms which are common to all tissues, such as fibroma, myxoma, sarcoma, tubercle, syphiloma, carcinoma, etc. To another we should restrict those nerve-growths made up largely of nerve-fibers or nerve-cells, and these only may properly be called *neuromata*. In addition there are mixed and transition forms. In the first group the tumors are commonly secondary and usually single. In neuromata the tumors are commonly multiple and the condition is often hereditary and may be congenital or may follow traumatism.

Of neuromata a number of varieties are encountered which have received descriptive names. Those made up of ganglion-like cells are called *ganglion or cellular neuromata*. When the nerve-fiber elements are present the term *fibrillar neuroma* is used, and this is further divided into the *myelinic* and *amyelinic* sorts, depending upon the presence or absence of the myelin constituent. The adjectives *terminal, central, peripheral, multiple, telangiectoid,* and *cirsoid* are merely descriptive of location or form.

Certain terminal neuromata which seem to be greatly enlarged Pacinian bodies constitute very painful tubercles under the skin, and to these the term *neuromata dolorosa* has been applied by Virchow.

The *multiple neuromata* constitute an important group. In some cases they number thousands, and vary in size from small peas to masses as large as a fist. They are located in three general ways: (1) They may appear along a single or along several nerve-trunks at somewhat regular intervals, like beads; (2) they may appear only on a single nerve and its branches, and (3) they may invade nearly every nerve in the body, including the sympathetic. In subcutaneous situations they appear like vascular, cirsoid dilatations, but present a different consistency and anatomical position, and are not modified by pressure on venous or arterial channels. They are most frequently acellular and devoid of myelin. While they may be sensitive and attended by neuralgic pains, they are usually unmarked by any sensory disturbance. As above indicated, such cases are sometimes of a hereditary nature.

Traumatic neuromata are rather common and their frequency after surgical operations has given rise to the term *amputation neuroma*. A nerve involved in scar-tissue by the healing process is likely to develop such a neuroma. In amputated limbs the nerve-stumps become clubbed, and upon examination there is found an increase in the nerve-fibers and axis-cylinders, which have a tendency to turn up and twist about in the small tumor, the size of which is relative to that of the nerve on which it develops. It appears to be but the thwarted natural effort of the central portion of a divided nerve to extend downward. Such traumatic neuromata are often exquisitely sensitive and may prevent the use of artificial limbs, besides causing much neuralgic suffering.

Neuromata in the limited sense of the term are benign growths, but occasionally after irritation or partial surgical removal sarcomatous conditions appear. From their number, and rarely from their position, as within the vertebral canal or cranium, or on the pneumogastric nerve, they may prove fatal. True neuromata are often associated with skin tumors and cutaneous brownish circumscribed pigmentations. Preble and Hektoen [1] from the clinical side divide cases of multiple neuromata into four groups which may be variously combined in a given individual: (1) tumors of the skin, (2) tumors of the nerves, (3) pigmentation of the skin, (4) functional disturbances.

Etiology.—In traumatic neuromata, and especially the amputation variety, the causation is readily understood. So is the action of irritation resulting in a fibrous proliferation which may strangle the nerve-fibrils, thrust them asunder, or locate a fibroid thickening on or within the nerve-sheath. The hereditary features of multiple neuromata do not explain the initial liability to this affection. They also appear in myxedematous, cretinoid, and phthisical conditions, and, strangely, are almost confined to males.

Symptoms.—Neuromata manifest themselves by local signs and by motor and sensory symptoms in the distribution field of the invaded nerves. When situated on the nerve-trunk they may present much the same features as a chronic neuritis or local nerve-injury. Hyperesthesia, paresthesia, and motor and trophic loss are likely to appear with the electrical formula of degeneration. If the neuroma is open to palpation, it presents usually a rounded, dense, nodular, more or less sensitive swelling. Owing to its attachment to the nerve it has considerable lateral movement, but resistance is encountered in the opposite direction. Pressure sometimes provokes neuralgic pains or tingling in the sensory distribution of the nerve. Multiple neuromata, however, may be quite insensitive and present no sensory, motor, or trophic disturbances. They are only to be detected by the often visible chains of nodular enlargements along the course of the nerves. Traumatic neuromata usually can be readily palpated in the region of scars and give a feeling of shot-like bodies which are usually very sensitive. The rare, painful tubercles constituting the neuromata dolorosa variety are readily palpated and give rise to pain and tingling in the definite nerve-area allied to their anatomical location. In some instances neuromata have caused muscular spasm in their neighborhood, or even at some distance, and epileptiform convulsions have been cured by their removal.

Diagnosis.—Neuromata are readily diagnosed when a palpable, sensitive enlargement is found on a nerve-trunk with sensory and motor disturbance below. In cases of multiple neuromata the diagnosis is apparent. When single and deeply situated, their presence may only be inferred by the slowness of the onset of symptoms and the very chronic course of the disease. At the same time neuritis and pressure upon the nerve from adjoining new growths must, if possible, be excluded. In this case, also, it will be impossible to decide whether the growth in the nerve belongs to the first group or is a true neuroma. Multiple neuromata, traumatic neuromata, neuromata dolorosa, and neuromata occurring in myxedematous individuals are usually of the true and, hence, benign variety.

Prognosis.—Single neuromata are more likely to damage the nerve than the multiple variety. If the tumor is of some size and functional disturbance is absent, none is likely to develop, but if such symptoms occur, they are likely to increase. True neuromata are of slow development and present a long course. Malignant or specific growths in nerves present the same outlook as elsewhere.

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[1] "Amer. Jour. Med. Sciences," Jan., 1901.

Treatment.—The treatment of neuromata is practically surgical. They must be excised with as little damage to the nerve-trunk as possible. In the multiple forms surgical interference is hardly practicable except for isolated masses, or to relieve special nerves. If the tumor is confined to the nerve-sheath, it may be removed without much injury to the nerve-trunk, but if this is involved, the neuroma must be exsected and the ends of the nerve sutured. This is now accomplished even when several inches of the nerve are removed, by interposing pieces of nerves removed from animals, or by catgut bundles, bone tubes, etc. There is, however, a decided liability to recurrence of neuromata after surgical interference, due to the preëxisting tendency and the irritant conditions set up by operation and healing. Nerve-stretching is claimed to be more efficient in relieving the reflex spasms than exsection. The use of anodynes for the relief of the neuralgic pains that sometimes make life burdensome should never be resorted to if the neuroma can be surgically dealt with. Pressure on the nerve above the tumor sometimes gives temporary relief from pain.

CHAPTER III.

LESIONS OF SPECIAL SPINAL NERVES.

ANY spinal nerve may be singly injured by trauma or disease, or several neighboring nerves may be involved at the same time by local conditions. Such lesions give rise mainly to disturbance of sensation, motion, and trophic control, showing themselves in varying degree in the cutaneous and muscular distribution of the injured nerves. The changed electrical conditions and the modified muscle reflexes that are also present equally depend upon the extent and nature of the lesion. These common lesions are division, neuritis, degeneration, and new growths, which have been considered in general terms in the preceding chapter. Irritant lesions produce morbidly exaggerated functions, such as spasms, hyperesthesias, pain, and rarely hypertrophy, while destructive lesions are marked by conditions of deficit, anesthesia, paralysis or paresis, dystrophy, and atrophy. These are often blended in the same case, as by the partial division of a nerve, or by a neuritis affecting mainly the motor or sensory portions of the nerve. For the sake of brevity, and to avoid repetition, these various nerve-lesions will be described systematically: (1) As to commonly acting causes; (2) as to the resulting motor disability and deformity, and (3) as to the sensory disturbance. A lesion of a disabling degree will be understood to be in operation unless otherwise indicated. Lesser injuries will, of course, present relatively diminished symptoms. In some otherwise intractable neuralgias the posterior spinal roots have been divided surgically within the spinal column, the resulting anesthesia being insignificant unless at least three consecutive roots are cut.

THE CERVICAL AND BRACHIAL PLEXUSES.

The phrenic nerve, arising from the third, fourth, and fifth cervical nerves, is impaired: (1) By disease of the cervical vertebræ or of the meninges or of the cord, affecting its spinal nuclei or roots. The condition is then usually bilateral, and other muscles than the diaphragm commonly suffer. (2) In the neck penetrating wounds may reach this nerve, or new growths injure it. (3) In the thorax tumors may compress it and inflammations extend to it.

The resulting *motor loss* consists of inactivity of the diaphragm on the affected side, which fails to descend on inspiration, and the corresponding portion of the abdominal wall does not advance equally with that of the sound side. This is especially noticeable on deep inspiratory efforts. When both phrenics are involved, the breathing is of a costal sort, and any exertion quickly causes respiratory distress. Difficulty in expectoration, sneezing, defecation, and other abdominal expulsive efforts is also present.

The *sensory disturbance* is obscure and often overlooked or misconstrued as intercostal neuralgia, muscular rheumatism, etc.

The posterior thoracic in its long course from the fifth, sixth, and seventh cervical nerves to its distribution in the serratus magnus is often subjected to mechanical pressure from heavy objects carried on the shoulder and by muscular compression in severe exertion or continued labor, particularly in overhead work. Mowing and tailoring also furnish cases. Penetrating wounds occasionally involve it, and falls or blows on the back may injure it. It also suffers in association with other nerves as a part of spinal atrophies. From the usual traumatic character of the disease, men in active middle life are most commonly affected, and on the right side, as a rule.

It occasions *weakness* in all the movements of the upper extremity that depend upon the fixation of the scapula and impairs thoracic inspiratory expansion on the same side, but causes no absolute motor loss. The paralysis of the serratus causes a peculiar and characteristic deformity. Attempts to put the arm forward cause the posterior border of the scapula to widely wing out from the chest, so that a deep recess is formed behind the shoulder-blade. The upper portion of the bone moves outward and the lower angle toward the spine.

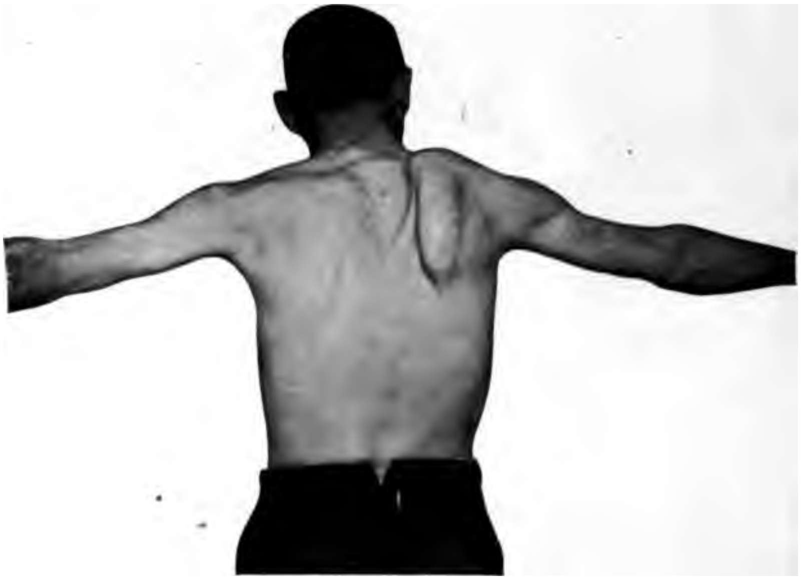


Fig. 102 A.—Partial paralysis of the right serratus magnus.

As the nerve is almost purely motor, the only *sensory disturbance* is neuralgic pain in the neck and shoulder in neuritic cases. The *prognosis* in injury to the posterior thoracic is comparatively less favorable than in other spinal nerves. A serratus paralysis is always of long duration and often permanent, even when there is every reason to believe

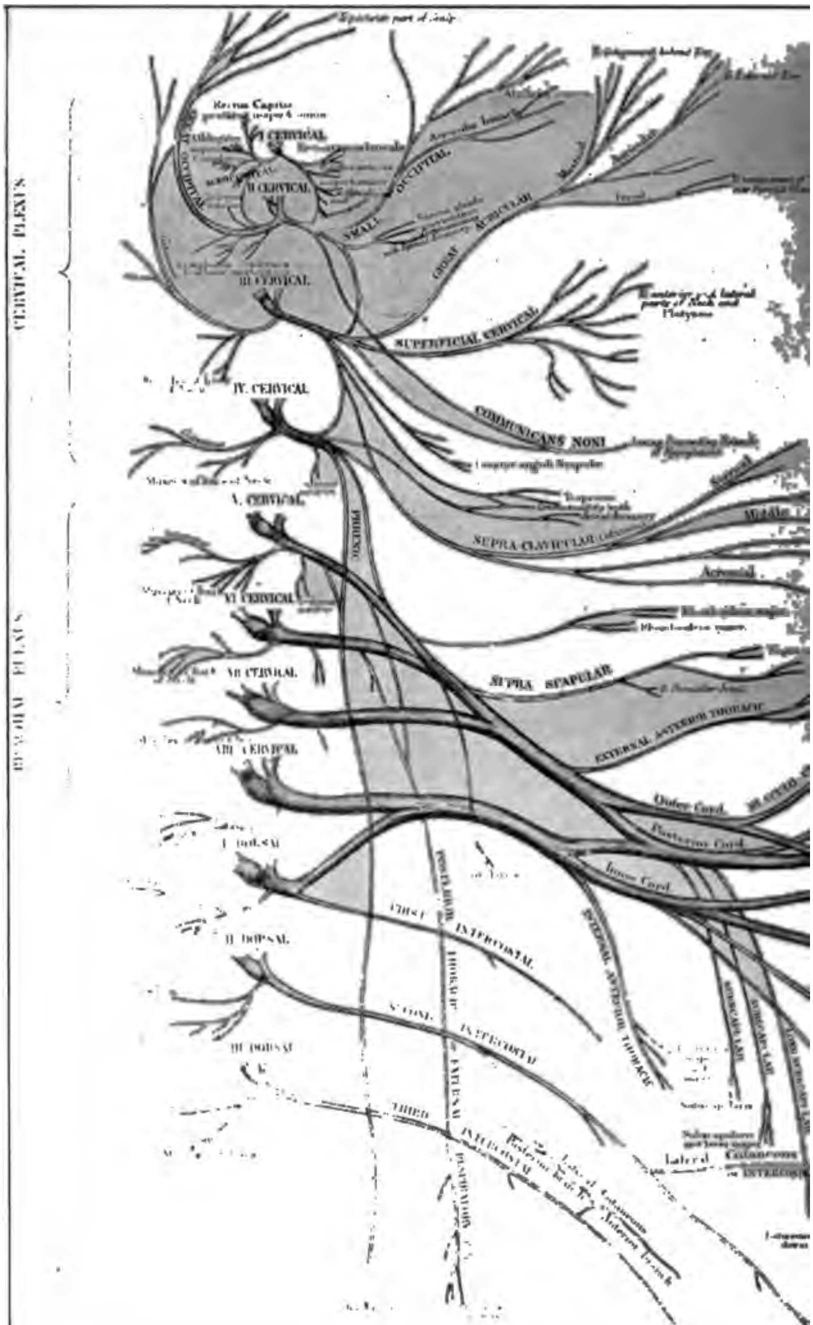
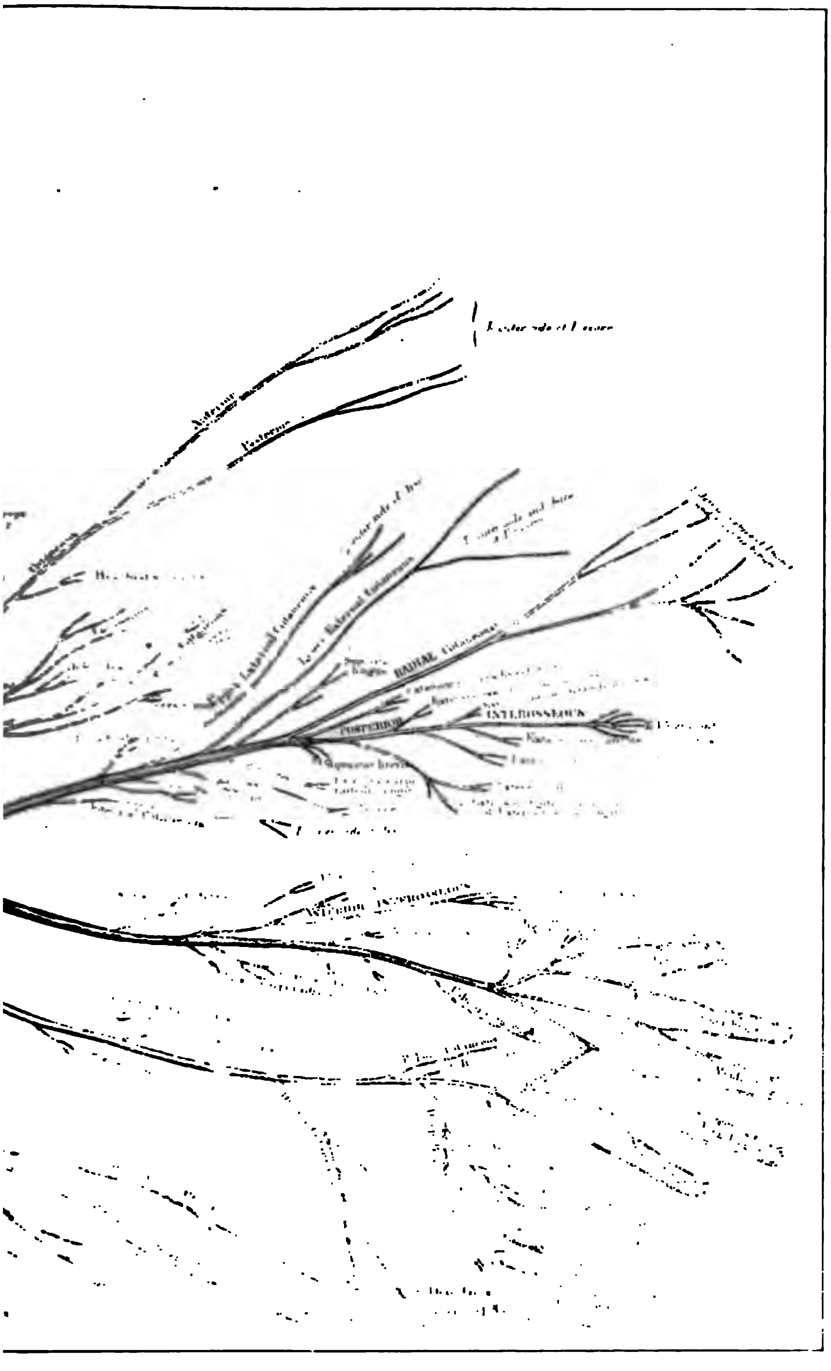


DIAGRAM SHOWING RELATIONS AND D

PLATE I.



AL AND BRACHIAL NERVES (FLOWER).

that the condition has arisen from a simple pressure neuritis. In cases not open to nerve suture Tubby[1] has suggested and successfully practised the ingenious operation of substituting a portion of the pectoralis major muscle, which is dissected from its humeral implantation, split up, and inserted into the digitation of the serratus.

The suprascapular nerve arises from the fifth and sixth cervical nerves. It may suffer alone in shoulder dislocations. The supra- and infraspinati are *paralyzed* and the scapula becomes subcutaneous by their atrophy. The arm can not rotate outward at the shoulder, and there is a general lack of balance with weakness in the movements of the member. Carrying the hand from within outward, as in writing, is rendered especially uncertain and difficult. *Anesthesia* over the outer portion of the scapula and the posterior portion of the deltoid is often present. Usually the suprascapular nerve and the circumflex are conjointly injured.

The circumflex nerve, arising from the fifth, sixth, and seventh cervical nerves, descends in the posterior cord of the brachial plexus, which it leaves to pass outward under the subscapular muscle, winds around the humerus, and is distributed to the teres minor and the deltoid. It also supplies the shoulder-joint. It furnishes sensation to the skin in a chevron-shaped area over the lower two-thirds of the deltoid. From its exposed position on the neck of the humerus and in the axilla it is often injured by shoulder dislocations, by arthritis, by crutch pressure, and by falls or blows on the shoulder. Injury of the circumflex causes *loss of action* on the part of the deltoid, and all attempts fail at lateral extension of the arm from the body. The loss of the teres minor action is insignificant.

Owing to the deltoid atrophy the acromion is uncovered and the shoulder rendered pointed and angular. The head of the humerus can readily be felt from the lateral aspect. In some instances it drops from its socket, leaving a deep furrow under the acromion. The nutrition of the joint also suffers and arthritis is likely to develop, limiting the range of joint motion. When the arm is passively moved, the scapula does not follow it unless joint disease is also present. An initial arthritis, by involving the articular branches, may spread to the circumflex and disable the deltoid.

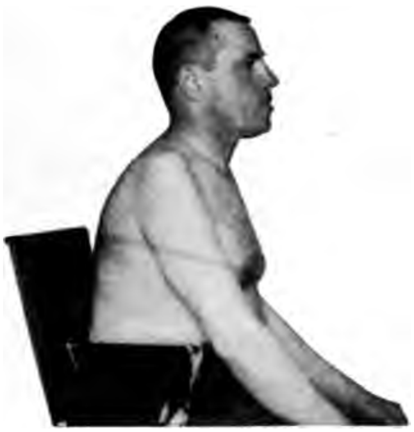


Fig. 103.—Atrophy of the deltoid and deformity of the shoulder in paralysis of the circumflex nerve.

Anesthesia in the distribution field of the circumflex over the lower two-thirds of the deltoid is usually present.

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[1] "Br. Med. Jour.," Oct. 9, 1904.

nerve is affected in the musculospiral groove, the usual location, the branches to the triceps escape and elbow extension is preserved. In lesions at or below the lower third of the humerus the branch to the supinator longus escapes, as it does in systemic affections. The wrist can not be extended and wrist drop is produced. The fingers are semiflexed and can only be extended by the action of the interossei—which are supplied by the ulnar—after the first phalanges are passively extended on the metacarpals. The thumb lacks extension movements, and those of the fingers progressively diminish from the index to the little finger. Frequently on the back of the carpus there develops a synovial tumor due to the overcarpal flexion, the inadequate support of the extensor tendons, and perhaps, in part, to the implication of the articular branches of the nerve. This constitutes a dense, painless elevation that exaggerates the wrist-drop deformity. Unbalanced by the extensor paralysis the flexors are weakened so that the hand-grasp is reduced in strength more than half. The muscular wasting shows most on the dorsal surface of the forearm. In those cases that involve the long supinator a very striking loss of contour is presented. The triceps may also show diminished proportions.

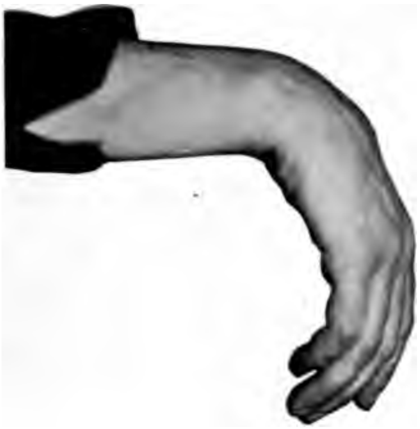

Fig. 105.—Dropped wrist from musculospiral palsy, showing retrocarpal tumor.

Sensory disturbance, in comparison with the extent of the paralysis, is very slight. Reference to the diagrams of cutaneous sensation (Figs. 15 and 16) will give an idea of the average space supplied by the radial branch of the musculospiral, but it varies widely in different individuals. The close relation with the median and ulnar also obscures the outlines of the field of disturbed sensation. One of the usual distributions of these nerves is indicated in figure 106. Often only prickling or slight numbness is felt in the tips of the thumb and index finger; in other cases the anesthesia is complete and sharply limited. In neuritic cases there is often complaint of constant pain in the wrist and carpal joints, which may be slightly swollen.

It is particularly in the treatment of dropped wrist that the great value of maintaining a proper position of the articulations may be emphasized. The tendency to carry the arm in a flexed attitude and the unopposed action of the flexors and pronators of the wrist give the carpal articulations a vicious position that is often difficult to overcome, and upon convalescence mechanically defeats the returning strength of the extensors. The use of a carefully padded and loosely applied anterior splint to maintain the wrist and fingers in line with the forearm hastens recovery in recent and in protracted cases.

The musculospiral nerve is the most frequently injured nerve in the arm, perhaps in the body. Arising from the posterior brachial cord, and arising originally from the fifth, sixth, seventh, and eighth cervical nerves or in some cases from the sixth, seventh, and eighth cervical and the first dorsal, it winds around the humerus in the musculospiral groove under the triceps, where it is subject to muscular compression and external violence or pressure. It supplies all the extensors of the elbow, wrist, and fingers, both the supinators, and through its radial branch the skin on the dorsal surface of the thumb and two radial fingers, and the posterior radial border of the hand. It also furnishes articular filaments to the wrist and carpal joints. By cutaneous branches given off above those to the triceps it supplies the skin in an area extending from the wrist in a narrow but widening strip up the dorsum of the forearm, and over the outer aspect of the arm as high as the insertion of the deltoid. These branches, however, are seldom involved in a musculospiral palsy. This nerve is injured in a variety of ways, and is especially involved in systemic states, such as lead poisoning. In these latter conditions it is interesting to note that the supinator longus, which is an active flexor of the elbow, does not participate. From its exposed position in the axilla, crutch pressure and dislocation of the humerus frequently affect it; lower down on the shaft of the humerus it is injured by fractures, nipped by callus, and subject to contusions from blows. Here it is frequently compressed injuriously by constricting cords about the arm, sometimes by violent action of the triceps, often by pressure during sleep with the arm under the body or resting on some hard object, as a chair-back, door-step, or bench. Such *sleep palsy* is sometimes presented after the prolonged stupor of drunkenness, surgical anesthesia, or narcotism. Direct blows to the arm may also produce musculospiral palsy, and cold is often accredited as a cause.

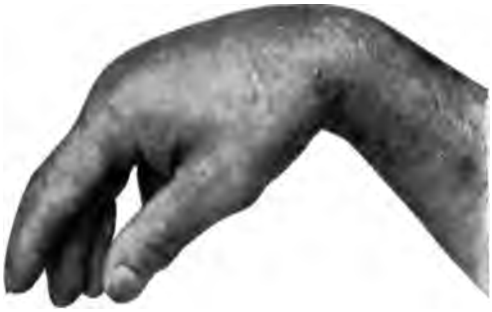

Fig. 104.—Characteristic position of hand in musculospiral palsy.

The *motor symptoms* of disease of the musculospiral nerve are extensive, interesting, and characteristic. The elbow and wrist can not be extended, and the long extensors of all the digits and the supinators of the hand are inactive after a lesion near the armpit. When the unusually extensive distribution of the ulnar and radial nerves. It is most pronounced on the volar surface of the index, but may extend over the area shown in figure 107.

The ulnar nerve originates from the lowest cervical and the first dorsal nerves, and supplies in the forearm the ulnar flexor of the wrist, the two inner divisions of the deep flexor of the fingers, and all the small muscles of the hand except those innervated by the median—namely, the dorsal and palmar interossei, the muscles of the hypothenar eminence, and the third and fourth lumbricales. It also supplies the adductor and one-half of the short flexor of the thumb. Its cutaneous branches supply the ulnar border of the hand, front and back, including all of the little finger, most of the ring finger, and a varying portion of the middle finger, largest on its dorsum.

The ulnar is (1) rarely injured above the elbow, excepting as a part of a more general injury to the brachial plexus. (2) At the elbow its exposed position behind the inner condyle and its superficial course through the forearm and at the wrist lay it liable to pressure neuritis and injury from wounds of all sorts. Even long-continued extreme flexion of the elbow-joint, as in sleep, in one predisposed, may suffice to induce a neuritis at this point. Occupations which necessitate continuous leaning on the elbow are also said to be active causes, but are certainly infrequent in this country. (3) Cuts at the wrist with various tools or from broken glass frequently divide it.

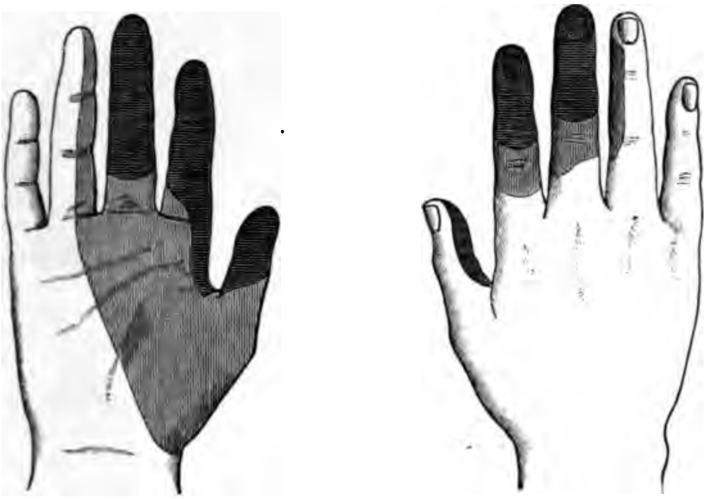

Fig. 107.—Showing areas of sensory loss in injuries of the median nerve (Bowlby).

Motor Symptoms.—Ulnar paralysis constitutes a serious disability of the hand. Flexion at the metacarpophalangeal joints and extension of the second and third phalanges, which are dependent on the interossei and lumbricales, are lost. The wrist can not be actively flexed to the ulnar side, and the thumb is rotated toward the palm by the abductor and opponens and can not be adducted. The fingers lose all lateral motion.

The distortion and deformity that result are pronounced and characteristic. There is overextension at the metacarpophalangeal joints, which makes the head of the metacarpal bones prominent in the hollowed palm. The unopposed flexors "claw" the second and third joints, and with the strongly-acting common extensor increase the deformity. This is least marked in the index and middle fingers, which do not lose their lumbrical muscles. All the interosseous spaces are emptied, and the fifth metacarpal is left entirely subcutaneous. In place of the hypothenar prominence there is a deep hollow. The unaffected muscles of the ball of the thumb stand out prominently in contrast with the skeleton-like-hand. The loss of sensation is confined to the indicated area of cutaneous distribution, but is only complete in the little finger. Figure 108 shows its distribution and degree.

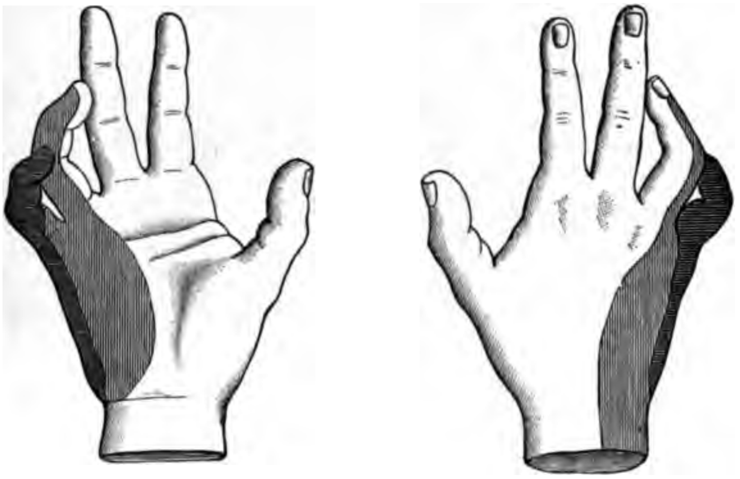

Fig. 108.—Showing sensory loss and ordinary position in injuries of the ulnar nerve (Bowlby).

COMBINED PALSIES OF THE NERVES OF THE ARM.

It is common for several nerves of the upper extremity to be injured at the same time, causing combined palsies presenting symptoms of corresponding extent. The circumflex, suprascapular, and musculocutaneous may be affected simultaneously by disease of the fifth and sixth cervical nerves, from which they arise. This is usually caused by forcibly dragging the arm downward or upward, lacerating the anterior nerve-roots[1] and may occur at birth. In this lesion sensory disturbance is usually absent. A new growth or injury at a point between the scaleni muscles opposite the sixth cervical vertebra may affect both spinal nerves. At this point Erb found they could be simultaneously stimulated by electricity. The muscles involved are the deltoid, spinati, biceps, and brachialis anticus, and the sensory disturbance corresponds to the cutaneous distribution of the three nerves in question. Kennedy[2] reports some such birth accident instances greatly benefited by surgical operation. A cicatricial condition was found at Erb's point above the

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[1] Huet, Duval, et Guillan, "Rev. Neurolog.," Dec. 15, 1906.

[2] "Br. Med. Jour.," Feb. 7, 1903.

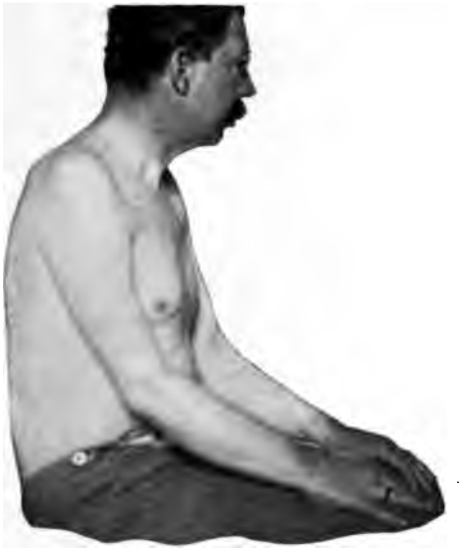

Fig. 109.—Radicular palsy affecting the spinati, deltoid, biceps, and brachialis anticus.

clavicle and outside the sternomastoid muscle; the nerves were resected and sutured with comparatively early restoration of motor function. Tubby[1] has remedied the defective biceps by utilizing the outer portion of the triceps, which is freed from its implantation into the olecranon and mattressed into the bicipital remnant.

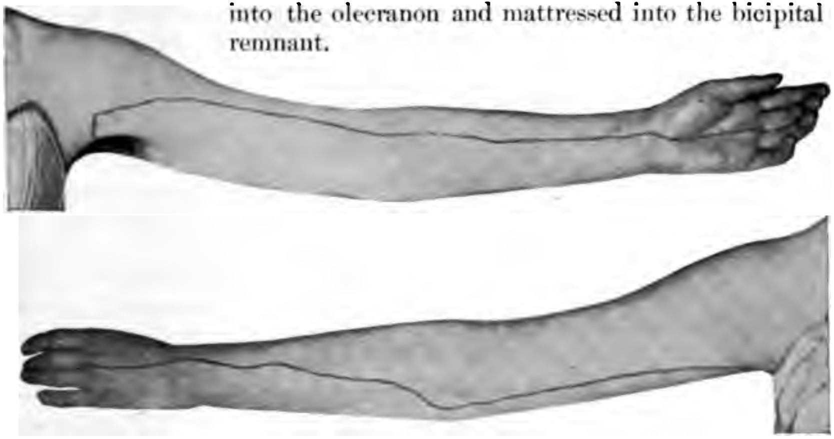

Fig. 110.—Brachial plexus injury. Area of anesthesia below black line.

Injuries to the brachial plexus usually involve more than one nerve in the arm. Nerve injury arising from dislocations of the humerus and from strains on the arm usually implicate several nerves. Thus the median, ulnar, and musculospiral may be injured together, or a single nerve may suffer. In figure 110 is shown a case suffering from a wrench of the arm received in alighting from a moving street-car. The ulnar, internal cutaneous, nerve of Wrisberg, and intercosto-humeral were simultaneously injured. The anesthetic area could be distinctly marked out. A similar combination of nerve-lesions occurs in birth-palsies from pulling on the arm or using a hook over the neck or in the axilla. Figure 111 shows the deformity in such a case: in a breech presentation attempts were made to draw the arm down from the side of the head and resulted in injury to the median, ulnar, and musculospiral.

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[1] Ibid., Oct. 17, 1903.

Fractures of the humerus may involve the ulnar with the musculospiral. Fractures in the forearm may injure the radial and ulnar nerves, which, also, may be simultaneously implicated in wounds at the wrist.

In an ascending neuritis other associated nerves are likely to be affected if the inflammation reaches the plexus (neuritis migrans). This most frequently results from infected wounds. A neuritis traveling up the median will first invade the ulnar and musculocutaneous, and then the other nerves arising from the brachial plexus. The order in which the symptoms arise is the key to diagnosis.

Disease of the cervical vertebræ, as Pott's disease; of the meninges, as cervical pachymeningitis; or disease of the cord, as syringomyelia, may involve the brachial nerves in varying groups.

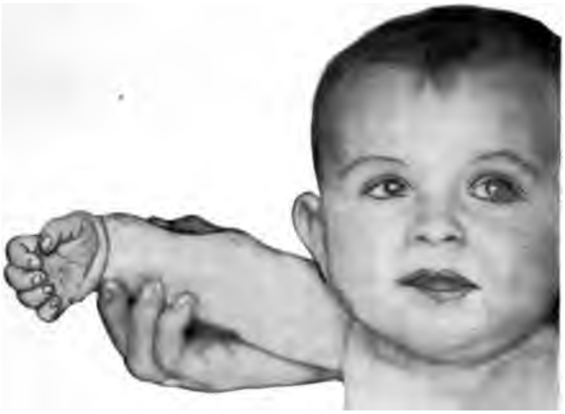

Fig. 111.—Brachial palsy from birth-injury, due to traction in axilla.

Neuritis of the brachial plexus is a rather common condition that is usually mistaken for a neuralgia, angina pectoris, or a rheumatic trouble in the shoulder and arm. According to Gowers, who first carefully described it, brachial plexus neuritis is analogous to sciatic neuritis in the lower extremity.

Causes.—This disease of the brachial plexus is encountered mainly after middle life, and especially in rheumatic and gouty subjects. In the majority of cases there is a history of attacks of lumbago or sciatica and a strongly marked lithemic heredity. Women suffer more frequently than men.

Symptoms.—*Pain* is the first and constant complaint. It is often of rather sudden onset and is likely to come in paroxysms, especially at night, becoming more continuous, but still with exacerbations. *Motion* that stretches the plexus, such as elevating the arm, is sure to provoke it, and any considerable use of the member is inhibited by the pain to which it gives rise. The pain may begin first in the region of the plexus, in the clavicular hollows, or under the scapula, but soon involves the entire arm, runs up into the neck, and frequently affects the side of the chest.

Upon manipulation one or all of the nerves in the arm will be found *tender* to the touch, and this *tenderness* can be traced through the axilla, under the clavicle, and into the scaleni muscles at the side of the neck. There is often tenderness over the scapula in the distribution of the suprascapular nerve. The skin over the arm is usually hypersensitive, and the constant dragging ache in the shoulder and arm is almost unendurable. The patient is constantly trying to find an easier position for the extremity, but without success. The tender *muscles* become flabby and wasted, and sometimes show slight degenerative reaction to electricity in certain groups. *Dermal,* epithelial, and vasomotor disturbances are not infrequent. The reflexes are diminished, rarely exaggerated.

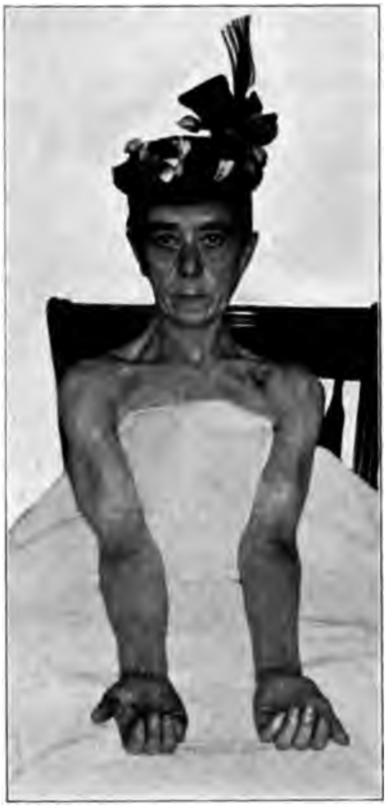

Fig. 112.—Neuritis of the brachial plexus on left side with wasting of arm and forearm.

Diagnosis.—The diagnosis rests on the continuous character of the pain, the tenderness of nerve-trunks, plexus, and roots, and the evidences of the neuritic process in the dermal changes, the vasomotor failure and the tendency to, if not the presence of, muscular atrophy. The presence of joint disease in old, gouty, and rheumatic patients may mislead as to the order of events, for the joint disturbance may be induced by the neuritis, or *vice versâ*. The history must determine the point.

Prognosis.—Brachial plexus neuritis is always a protracted malady, requiring from three to eighteen months, or more. In old arthritic individuals it is especially inveterate, but usually terminates in recovery. Relapses are likely to take place, and some slight disability in the way of pain or weakness is likely to persist permanently. The long maintenance of flexed positions, with the double tendency to joint disturbance furnished by the rheumatic element and the nerve-lesion, frequently result in a limitation of the range of motion at the shoulder and elbow. The wrist and smaller joints may also be affected, and the use of the hand considerably impaired.

Treatment.—The treatment is that for neuritis with especial attention to the gouty state. Repeated counterirritation on the side of the neck, in the clavicular fossæ, and on the inner aspect of the arm with blisters or the Paquelin cautery may be used from the first. The arm should be immobilized, except for the mildest daily passive movements, slight massage, and electricity when the tenderness and pain permit.

NERVES OF THE TRUNK.

The individual nerves supplied to the trunk by the thoracic and two upper lumbar pairs are rarely singly involved. A vertebral lesion or a wound may so affect them, but the motor loss is hardly appreciable unless several suffer simultaneously. The neuritic pain arising from Pott's disease and the girdling sensation in this malady and in tabes dorsalis have great value from a diagnostic standpoint. There is also some truth in the lay apprehension that herpes zoster may be fatal if it completely girdles the trunk, as in such instances it almost always arises from some implication of the nerve-roots by spondylitis, malignant growths, or other serious local mischief. Though irritation of a nerve may cause a localized herpetic eruption, in true zoster the lesion is an inflammatory disturbance of the posterior root ganglia (see page 32[illegible]).

THE NERVES OF THE LOWER EXTREMITY.

The nerves of the lower extremity are much less frequently diseased than those of the arm, but are subject to special accidents by pressure on the lumbar and sacral plexuses within the body, as from parturition and abdominal and pelvic growths. Psoas abscess and inflammations of the pelvic viscera may also invade these plexuses.

The anterior crural nerve may be affected (1) within the abdomen, when the iliacus is affected and flexion of the thigh on the body is weakened, or (2) below Poupart's ligament. After a *paralyzing lesion* the extensors of the knee are inactive and wasted and the knee-jerk is abolished. Difficulty is experienced in advancing the foot, especially in mounting stairs. *Anesthesia* extends from the groin to the inner side of the foot, involving the entire extremity except the buttock and a narrow strip down the back of the thigh, which below the knee spreads over the outer side of the calf and embraces the foot excepting on the inner margin (see diagrams, pp. 52 and 53). This nerve was observed to be affected seventeen times in 1000 patients in the lying-in hospital of Copenhagen, in all subsequent to parturition.[1]

The external cutaneous branch of the anterior crural supplying the outer side of the thigh seems especially subject to sensory disturbances. In gout, alcoholism, tobacco excesses, and sometimes as a lingering feature in multiple neuritis, this area is sometimes exquisitely hyperesthetic, so that the patient finds it inconvenient even to allow the weight of the trousers upon the skin, or is conscious of an acute tingling when he passes his hand over the thigh, or complains of various persistent paresthetic sensory disturbances. This condition is called *meralgia paresthetica* and may be due to defective return circulation associated with varicose veins and hemorrhoids. It is usually a neuritis and the nerve has been found thickened at the point where it crosses the pelvic brim.

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[1] "Centralbl. f. Gynäk.," No. 25, 1901.

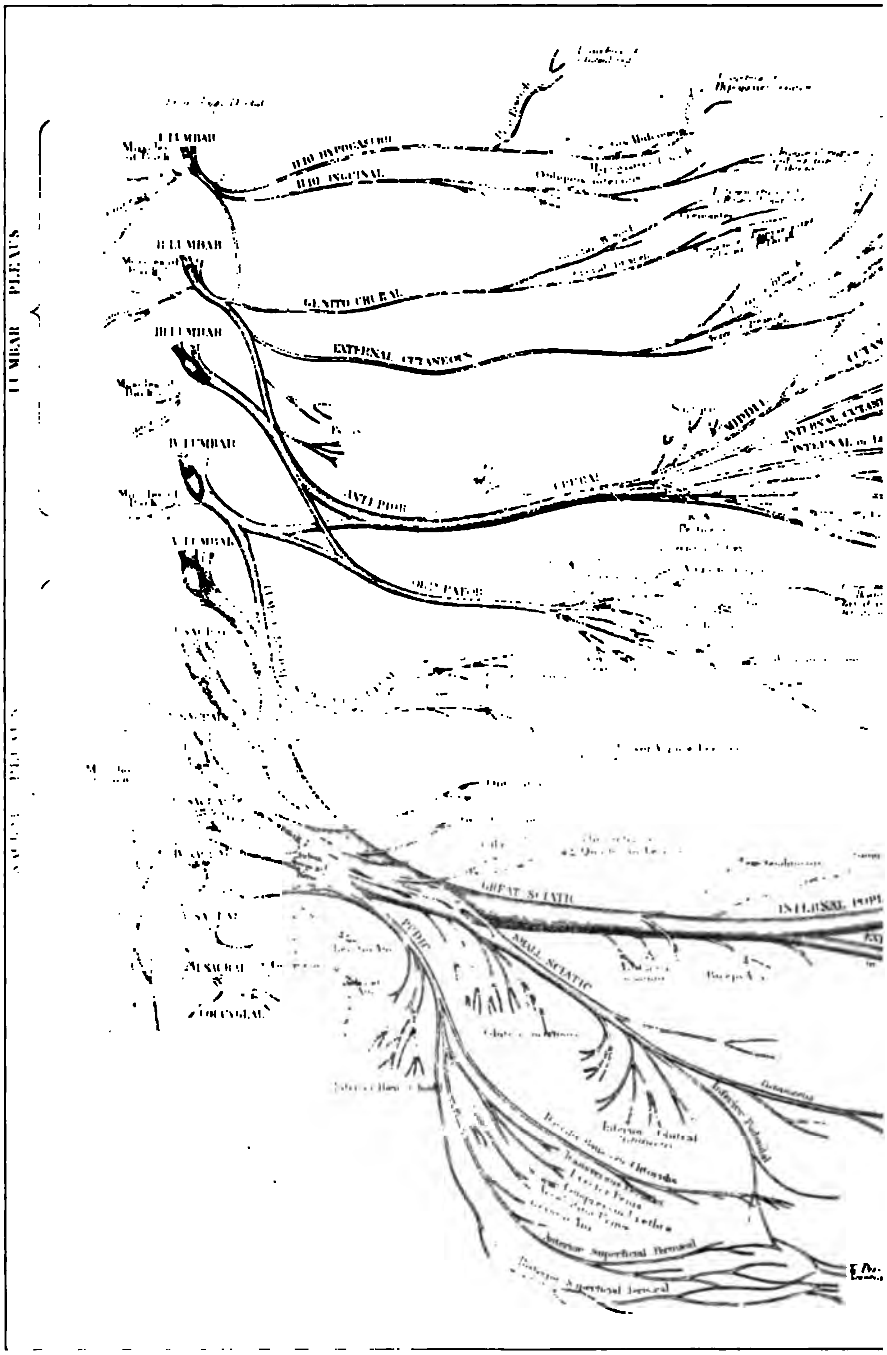

DIAGRAM SHOWING RELATIONS AND DIS

BAR AND SACRAL NERVES (FLOWER).

The obturator nerve has the same origin as the anterior crural and supplies mainly the adductors of the thigh and the hip- and knee-joints. Its *paralysis* interferes with movements that require approximation of the knees and impairs outward rotation of the thigh through weakness of the pectineus and external obturator. There is wasting on the inner aspect of the thigh. The *sensory disturbance* is confined to the hip- and knee-joints, which may also display trophic disturbance. The knee pain in morbus coxæ is a familiar complaint due to irritation of this nerve.

The superior gluteal nerve supplies the gluteus medius and minimus and the tensor vaginæ femoris. Its injury interferes with abduction, outward rotation, and circumduction of the thigh.

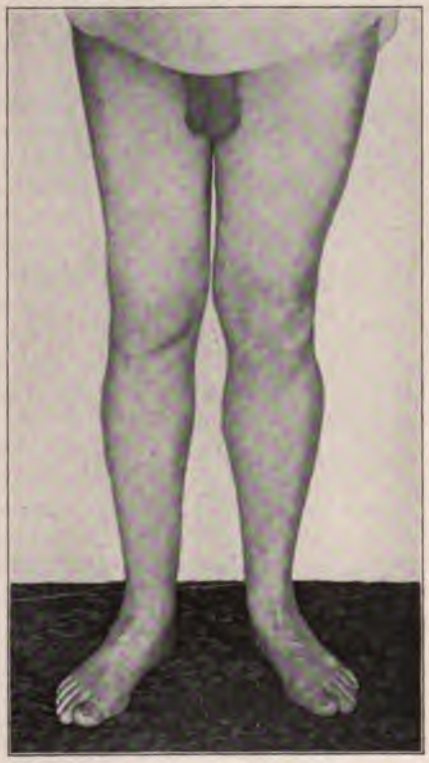

Fig. 113.—Paralysis of anterior crural; wasting of right thigh.

The great sciatic nerve is diseased singly more frequently than any other nerve in the lower extremity. Its size and exposed position within the pelvis, at the sciatic notch, and behind the neck of the femur, and the exposed position of its branches, notably the external popliteal as it turns around the fibula just below the knee and the plantar branches in the sole of the foot, lay it especially liable to accidents. It supplies the flexors of the knee, which also assist in maintaining extension of the body on the hip, and all the muscles below the knee. In spite of its size and extensive muscular distribution, *paralysis* of the sciatic nerve is not so disabling as that of the anterior crural. The hip-joint can be fairly controlled by the muscles that escape, and the knee is kept in extension through the anterior crural. The whole extremity is then thrown forward *en masse* and used as a peg-leg, as in the hemiplegic gait (see p. 210), but without rigidity.

The *anesthesia* that results from palsy of the great sciatic occupies a narrow strip from the gluteal fold to the ham, where it spreads, embracing the outer side of the leg as far forward as the tibial crest, and all the ankle and foot except on the inner side. The wasting is marked on the back of the thigh and below the knee; the limb may be reduced to the last degree. Perforating ulcers on the sole of the foot, particularly at the ball of the great toe, are likely to develop.

The external division of the sciatic, the *external popliteal* or *peroneal nerve*, occupies an exposed position (1) in the ham and (2) below the knee on the outside of the neck of the fibula. It supplies the extensors of the foot, the long extensors of the toes and the peronei, and is analogous to the lower portion of the musculospiral in the arm. Its paralysis causes foot-drop and toe-drop. The muscular fullness on the anterior and outer aspect of the leg is lost; the uncovered crest of the tibia is extremely prominent. A tendency to equinovarus is produced that is often rendered a permanent deformity by contracture in the calf-muscles. The outer half of the front of the leg and the dorsum of the foot are anesthetic.

The inner division of the sciatic, the *internal popliteal*, is analogous to the median and ulnar in the upper extremity. It supplies all the calf-muscles, the long flexors of the toes, and all the small muscles of the foot except the short extensor of the toes. It divides into the internal and external plantar nerves, which are sometimes singly diseased. When the *internal plantar*, which is analogous to the median, is alone divided, we have paralysis of the short flexor of the toes, the plantar muscles of the great toe except the adductor, and the two inner lumbricales. The big toe becomes overextended at the first joint and flexed at the second, producing the deformity called *hammer-toe*. There is anesthesia on the inner portion of the plantar surface, embracing also the plantar surface of three and a half toes.

When the *external plantar*, the analogue of the ulnar, is divided, the muscles of the little toe, the two outer lumbricales, the adductor hallucis, and all the interossei are paralyzed. All the toes assume the hammer-toe deformity, and the use of the foot is much weakened. Anesthesia is produced on the outer half of the sole and on the plantar surface of the little and one-half of the next toe. The dorsal surface of the foot and toes is supplied by the musculocutaneous and anterior tibial nerves from the peroneal.

When the internal popliteal is divided in the ham, in addition to the disability due to paralysis of both plantar nerves we have a loss of inward rotation of the flexed leg, due to paralysis of the popliteus muscle, and loss of power of extending the foot on the leg. By the overaction of the anterior leg-muscles talipes calcaneus is produced. The arch of the foot at the same time becomes exaggerated; the toes are extended at the first and flexed at the other joints, producing the analogue of the combined ulnar and median deformities in the hand.

Metatarsalgia, or *Morton's disease*, probably consists of a pressure neuritis or neuralgia of some, usually one, of the digital branches of the plantar nerves between the heads of the metatarsal bones. It commonly arises from the use of too narrow boots, and can be remedied in some cases by a shoe of proper width snugly embracing the arch of the foot. In the sole a depression is fashioned to relieve pressure on the particular painful point. Excision of the metacarpal head or neurectomy have both been employed successfully in severe cases.

Sciatic Neuritis.—Neuritic and neuralgic conditions have been so confused under the term sciatica that it is best avoided. Persistent pain confined to the region of the great sciatic nerve and its distribution, with tenderness in the nerve-trunk, is almost always due to neuritis. Sciatic neuralgia, on the other hand, is a rare affection. Inflammation of the sciatic nerve, from its clinical importance, demands separate consideration.

Causes.—The causes of sciatic neuritis are those of neuritis elsewhere, and may be divided into those of a general and those of a local sort. In the first group we may name *adult age*, as sciatic neuritis is comparatively rare before twenty-one. *Males* are much more commonly affected than females, in the proportion of 8 to 1, according to Gibson.[1] *Gouty*, *rheumatic*, and *neurotic* individuals are especially liable to develop it. Among *systemic poisons* that favor its occurrence may be mentioned lead, diabetes, syphilis, typhoid fever, malaria, and grip. *Pelvic disease* and pelvic hyperemias seem also active in causing sciatic neuritis, often acting also as local causes by transmitting pressure or inflammation to the sacral plexus. It is most frequent in *wet* and *cold seasons* and among those whose *occupations* expose them to wet weather or extreme changes of temperature, such as stokers, puddlers, and laundresses. It may follow operations done in the lithotomy position.

Among the numerous *local causes cold* is usually considered most important. Sitting on damp ground or on cold stones, standing in water, or wearing wet clothing or shoes. *Compression* of the nerve in sitting or by vigorous action of the leg-flexors and rarely *contusions* from blows below the sciatic notch may induce it. Bony thickening at the notch and syphilitic deposits at this site have occasioned a sciatic neuritis. In quite a proportion of cases there is an antecedent lumbago. The downward extension of the process in the muscles and fascia involves the sciatic nerve at the notch. All sorts of *pelvic tumors* and even *constipation* are capable of exercising such pressure on the sacral plexus that a sciatic neuritis follows. The nerve is sometimes injured in this position by the *obstetrical forceps* or through compression by the *gravid uterus* and *fetal head*. *Spinal disease* and new growths within the spinal canal may give rise to neuritis by affecting the nerve-roots in the cauda equina or at the vertebral foramina. *Excessive fatigue* of the legs, as in the use of the sewing-machine, and the pressure of *varices* in the extremity or in the pelvis or about the nerve-trunk may occasion it.

Morbid Anatomy.—From rare autopsies and occasional operations the condition in the nerve is found to be a perineuritis, affecting also the adventitia. Secondarily the nerve-fibrils suffer. There is often an increased vascularity and redness of the nerve, and varicose dilatations in and about the nerve have been observed by some. The nerve is usually edematous and tumefied. In a word, we have to deal with an interstitial neuritis. Hunt[2] describes a perineural jelly-like, quite structureless deposit in the nerve sheath unaccompanied by inflammatory changes and probably of gouty or rheumatic relationship. It is usually most marked at the sciatic notch and in the middle of the thigh, but may be more widely diffused. A similar condition is sometimes found in the branches, particularly the external popliteal.

Symptoms.—The primal symptoms of sciatic neuritis are *pain* and *tenderness*. While both have a tendency to extend throughout the sciatic territory, at first, and often during the entire case, they are confined to the crural portion of the nerve and are most intense below the gluteal fold and in the upper half of the thigh directly in and over the nerve-trunk. The patient will often trace with his finger the seat of pain in a line that anatomically corresponds to the nerve itself. Below the knee the pain follows by preference the external popliteal nerve. The pain varies in degree, but is persistent in character, and even when slight is extremely wearing by its continuance. It is notably subject to exacerbations, which are quite likely to occur at night and seriously interfere with sleeping. Walking or any vigorous use of the limb is likely to increase the pain at once. Even after many hours of freedom from it, the pain may sometimes be promptly reëstablished by taking a few steps. In bed the patient holds the limb by preference in a semiflexed position at the hip and knee with the ankle extended. This obviates compression of the nerve by the muscles and avoids stretching the nerve by extension. If the nerve is stretched over the femoral neck, by extending the knee and flexing the thigh on the pelvis, pain is at once produced. These conditions give rise to a characteristic *attitude* and *gait*. The patient holds the knee semiflexed and inclines the body to the opposite side, throwing into prominence the hip on the side of the sciatic neuritis. This tends to keep the weight on the sound limb, but its long continuance may develop a well-marked scoliosis with the lumbar spine convex to the neuritis and a compensatory dorsal curvature convex to the sound side. After recovery from the neuritis this *sciatic scoliosis* usually disappears. The scoliosis occurs in the opposite sense in cases marked by spasms in the affected leg and thigh, which are due to involvement of the sacral plexus or the lumbar cord, and manifest especially in the distribution of the anterior crural nerve. The hip on the side of the neuritis is then approximated to the lower ribs, the opposite one made prominent; the lumbar curve is concave, the dorsal curve convex, on the side of the disease. This *homologous sciatic scoliosis* is likely to be permanent inasmuch as the condition upon which it develops is a chronic one and contractures develop to fix the deformity. In walking there is an evident intention to use the diseased limb as little as possible in order to avoid tension and muscular compression of the inflamed nerve.

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[1] "Lancet," London, 1893, No. 3633.

[2] "Am. Med.," Apr. 15, 1905.

Pressure made with the fingers along the trunk of the nerve and its branches develops tenderness, which is greatest in certain regions. These correspond to the *tender points* of Valleix. The most constant are the following: The *gluteal point* over the sciatic notch, *the trochanteric point* above the trochanter major, a *tract* corresponding to the nerve-trunk on the posterior aspect of the thigh, a *popliteal point* in the ham at the division of the nerve, a *fibular point* where the external popliteal is superficial to the neck of the fibula, and a point on the *dorsum* of the foot. Less frequently we find *lumbar points* just above the sacrum, an *iliac point* at the middle of the iliac crest, a *patellar point* over the knee-cap, points in the *calf*, points behind the *malleoli*, and *plantar points* in the sole of the foot.

It is at these points that the patient complains of the *paroxysms of pain* that commonly mark the course of the disease. They are often of a lancinating, boring, tearing, burning character that racks the patient and exceeds his power of description and endurance. They are usually provoked by use of the member, by an exposure to cold, or by some manipulation of the parts, but may come on spontaneously and even periodically, as in malaria. They often follow sleep, probably from pressure or a continued cramped position.

Cutaneous sensibility is often modified. Hyperesthesia in the sciatic area is the rule at first, but in prolonged or severe cases gives place to anesthesia. Often the hypersensory disturbance is confined to small areas which correspond fairly well to the painful points previously indicated. Various paresthesias are encountered, such as a feeling of cold, heat, prickling, fullness, formication, etc.

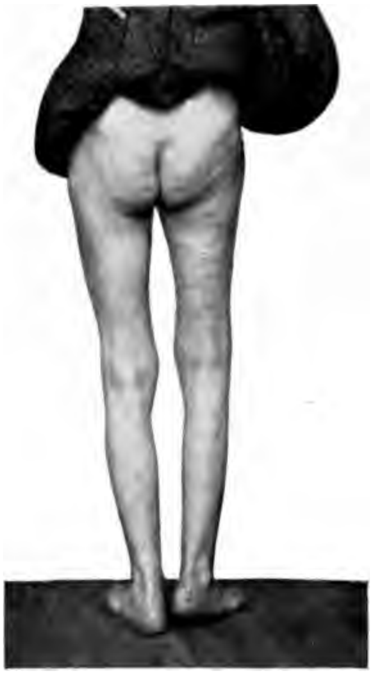

Fig. 114.—Sciatica of long duration on right side; wasting of buttock and leg, some contracture with tendency to stand on toe, elevation of hip, and scoliosis.

Motor disturbances are less constant and present much variation. They usually only appear in the graver cases. Sometimes the member is shaken in a spasmodic manner during the painful paroxysms, and painful cramps in the calf-muscles may aggravate the patient's suffering. *Reflexes:* The knee-jerk is rarely exaggerated, usually it is diminished, and the Achilles tendon reflex is almost always greatly reduced or absent. Gibson[1] notes that the cremasteric reflex is usually exaggerated and the plantar may be increased, but the toe-sign of Babinski is never present. If the muscles waste, fibrillar twitching is frequently observed, and there is paresis or paralysis. In the rare spasmodic cases already mentioned, in which the plexus or cord is involved, the entire limb may be drawn up in a painful and protracted spasm upon the slightest cutaneous irritation, even by a breath of air or the contact of clothing.

Involvement of the vasomotor and trophic functions of the nerve in severe and protracted cases renders the limb livid in color, cold to the touch, with a temperature reduced several degrees. The perspiration is usually reduced, but may be increased, and the dermal structures frequently suffer, as in neuritis elsewhere. Scaliness, herpetiform eruptions, erythema, and acne sometimes appear, and perforating ulcer has been encountered. The muscles waste and show the reaction of degeneration to electricity. The wasting is most noticeable in the leg and buttock. The enlarged nerve may sometimes be felt. An increase of cutaneous fat may mask the muscular wasting.

A double sciatic neuritis is almost invariably due to spinal disease or pelvic disorder, to the suspicion of which it should always give rise.

Diagnosis.—The diagnosis of a sciatic neuritis is not ordinarily difficult. It is based on persistent pain and upon the presence of the anatomically located tenderness. Neuralgia gives rise to fleeting pain without tenderness or with relatively very slight tenderness, but early in neuritis this combination may also obtain. Disease of the hip-joint is sometimes mistaken for sciatic neuritis. Here joint tenderness and fixation and the location of the pain, which does not correspond to the sciatic but to the obturator nerve, serve to differentiate the articular disease. Hysteria sometimes mimics sciatic neuritis. The onset of the disorder under emotional stress, the absence of trophic disturbance, and the presence of contractures and other stigmata of the neurosis distinguish it. When the neuritis has resulted in wasted muscles, cutaneous eruptions, etc., it can hardly fail of recognition.

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[1] "Edin. Med. Jour.," No. 9, 1901.

More difficult questions are those of the causation and location of the primal inflammation. In every instance a thorough investigation should be made of the pelvic organs and the spinal functions. If the neuritis is bilateral, the disease is almost surely above the sciatic notch. In tabes sciatic pains are very frequent, but their lancinating character and appearance in storms, with other symptoms and signs of the disease, should lead to their proper classification. By the injection of cocain into or close to the nerve at the sciatic notch the neuritic pain is inhibited, unless the trouble is higher up, and this measure, therefore, helps to locate it within or outside the pelvis. A history of syphilis, malaria, or climatic exposure sometimes tells the whole story.

Prognosis.—A pure sciatic neuritis is of good ultimate prognosis. If, however, it be due to pelvic or spinal disease, the outlook is modified for the worse by such conditions and in proportion to their gravity. As in any other neuritis, the probable duration must be estimated from the degree of severity of the neuritic process. The manageability of the patient has much to do with the prospects of a given case, as a great deal depends upon rest, and preferably rest in bed. Usually the disease lasts several months, and under conditions of use and irritation may extend over years or present numerous relapses. If nerve degeneration and muscular atrophy develop, from six to nine months will be required for recovery, though the pain may subside much sooner.

Treatment.—The general plan of treatment of a sciatic neuritis corresponds to that of any ordinary neuritis (p. 284), but certain local conditions require special attention. First of all and most important in the *early stage* is complete rest of the limb by absolute rest in bed. This can be rendered the more effective by the use of a long, well-padded splint, extending from the armpit to below the foot, where a cross-piece on the surface of the bed serves to maintain a proper position of the whole. To this splint the limb should be lightly secured in a comfortable position, slightly flexed at the hip and knee. The upper end of the splint is secured to the chest by webbing bands or a binder. Care must be taken not to constrict the limb at any point. Gouty, rheumatic, and other favoring conditions require appropriate attention, and, of course, pelvic and intestinal trouble must be properly treated. From the very first, counterirritation over the nerve-trunk should be employed. This may be in the form of sinapisms, small blisters, or preferably by the Paquelin cautery. Continuous use of hot poultices or the ice-bag is to be advised.

Internal medication is practically useless, though some favor mercury, others salicylates, and lately nitroglycerin, in $\frac{1}{100}$-grain doses every two or three hours, has been lauded. In the writer's experience it has had no value. Acupuncture over the course of the nerve serves the same purpose as any other form of counterirritation, and the same is true of injections of ether and other fluids. Gibson [1] advises puncturing the nerve-trunk itself with a good-sized needle in about five places, and reports, in 100 consecutive cases, 56 cured, 32 much improved, 10 improved, and 2 unimproved. The purpose of the nerve-puncture is to relieve the supposed edema within the sheath. The usefulness of the measure is, therefore, limited to early cases. For the same reason the needle should be used at the point of involvement, which is usually opposite the gluteal fold. When the nerve is thus pierced, a pain darts down its course. The needle is to be inserted at intervals of about an inch in the course of the nerve in the thigh, five times at a sitting, and repeated after two or three days. Negro [2] reports 113 rebellious cases in which vigorous pressure over the nerve at the most painful part resulted in recovery. He places the patient on his face and with all his force presses both thumbs upon the nerve, rolling it from side to side, for fifteen seconds. This is repeated after twenty minutes and it is then much less painful than at first. The patient is relieved of pain for several hours and is enabled to walk. It is recommended that this manipulation be done about six times at two-hour intervals every second or third day. This procedure would seem better calculated to cause than to cure a neuritis, but is certainly valuable. Vigorous massage of the nerve in early cases has always seemed, in the writer's experience, to increase the pain and intensify the trouble, but in late cases is of great benefit, particularly in building up the wasted muscles. Electricity is also extensively used by some. When the inflammation is recent and the pain severe a majority favor a broad, positive sponge-electrode over the nerve, with sufficient unbroken current to redden the skin. In old cases the vigorous faradization of the thigh- and leg-muscles, though painful, sometimes seems to be of benefit, perhaps by the mechanical muscular pressure thus induced. Stretching the sciatic over the neck of the femur by forcibly flexing the hip-joint with the knee fully extended is also useful in late stages.

In most cases sedatives are required. Morphin is the only certain anodyne, but its disadvantages in the face of a protracted disease should postpone its use to the last moment. The coal-tar derivatives have some effect, and cocain, $\frac{1}{10}$ of a grain injected near the nerve, acts very well. The best results will probably follow complete rest and vigorous counterirritation in the early stage; active massage, cutaneous stimulation, and free use of the limb in protracted cases.

Various springs and watering places have gained a reputation in the treatment of sciatic neuritis. All furnish hot baths and much rubbing or rude massage. The enforced idleness, abundant excretions due to drinking the water, frequent baths, and manipulations are the factors of their success. In early cases such a course is often clearly detrimental.

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[1] "Lancet," London, 1893, No. 3633.

[2] "Bulletin Méd. de Paris," Jan. 22, 1896.

CHAPTER IV.

MULTIPLE NEURITIS.

UNDER the terms *multiple neuritis, multiple peripheral neuritis, polyneuritis, beri-beri, kakké,* etc., are embraced a number of conditions which have groups of symptoms mainly referable to disturbed functions of the peripheral nerves. The old-time distinction between central and peripheral organs and the clinical features of these cases directed attention mainly to the nerve-trunks and endings. In view of our present knowledge of the neuron unit, supported by numerous observations showing spinal nuclear involvement and even cerebral cortical disturbance in multiple neuritis, the term peripheral must be dropped. By multiple neuritis or polyneuritis is here meant a malady in which the anatomical lesions open to our present means of investigation are usually more pronounced in the nerves than in the central organs, and commonly consist of degeneration of the axis-cylinder process. Cases in which no histological change is found can not always be excluded, as many of the numerous poisons giving rise to the disease may, so far as we can detect, sometimes produce dynamic modifications alone. It is also allowable to suppose that the early effects of such poisons are perturbative of nerve-cell activity, to which the degeneration in the distant axis-cylinder process is secondary, and that if the poisoning be slight in degree, or the investigation be made before the secondary results are developed, no change whatever will be presented. Finer methods, however, are daily displacing post-mortem appearances which were formerly considered normal, and the dynamic cases are being steadily brought within the domain of the morphologically abnormal. It is likely that polyneuritis were better classed as a general disease of the nervous system. The preponderance of neuritic conditions has dictated its description in this connection.

Etiology.—Polyneuritis is the result of a systemic poisoning or of conditions which entail depraved nutrition, such as pernicious anemia, or of both. *The toxic substances* which are capable of producing a multiple neuritis are most commonly alcohol and lead; less frequently arsenic, mercury, silver, phosphorus, sulphid of carbon, oxid of carbon, and ergot. In Manchester, England, an epidemic of multiple neuritis was traced to arsenic in the beer.[1] Certain *autotoxic* substances may be developed that have the same pathogenic power, as in diabetes, nephritis, and intestinal disturbance.

The infections furnish another group of causative agents. Most of the infectious fevers—typhoid, typhus, the exanthemata, erysipelas, puerperal infection, acute rheumatism, influenza, and, above all, diphtheria—are occasionally followed by a multiple neuritis, which usually appears during convalescence. Even whooping-cough has been complicated by multiple neuritis.[2] In rare instances the neuritic disturbance appears early in these infectious diseases, and again rarely it may follow them after a considerable period of time, even after several months have intervened.

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[1] J. S. Bury, "Br. Med. Jour.," Dec. 1, 1900.

[2] Eshner, "Jour. Am. Med. Assoc.," Jan. 10, 1903.

Syphilis and *tuberculosis* have both been cited as causal of polyneuritis. Undoubtedly they both predispose to it by the systemic depression they occasion, and both are capable of producing a local neuritis by their specific proliferations. The anesthetic form of *leprosy* is frequently marked by a polyneuritis which is actually a peripheral disease and due to the invasion of the nerve-trunks by the bacillus lepræ. *Malaria* can undoubtedly produce it. Eisenlohr, in Homburg, and Graeme Hammond, in Connecticut, have reported small epidemics of the disease, which also in the form of beri-beri is common in certain parts of Asia, South America, and on shipboard during long voyages.

Cachexias of all sorts, but especially those associated with cancer, are likely to develop polyneuritis; usually, however, combined with changes in the spinal cord. *Old age,* and especially old age marked by extreme arteriosclerosis and atheroma, may be the occasion of the disease by failure of nutrition both in the trophic spinal apparatus and in the peripheral nerve-trunks. Multiple neuritis occurs infrequently before adult *age.* In children it is usually due to infection, especially diphtheria, measles, and influenza. Much more rarely it is caused in childhood by lead or arsenic. Arsenic has caused polyneuritis in some cases of chorea where it has been too freely used. After adult age is reached all forms are comparatively common. Alcoholic polyneuritis is most frequent between thirty and forty. The senile and atheromatous forms appear only after sixty. Women furnish the greater proportion of alcoholic cases; men, of the rheumatic and toxemic variety. *Occupation* plays a large part. Painters, plumbers, type-setters, rubber-workers, match-factory employees and employees in white-lead factories, and lead-miners are especially exposed. Various other pursuits requiring the use of lead, arsenic, and mercury, or the handling of spirituous liquors, the last by indulgence, furnish many cases. Very often more than one cause is operative, as when tuberculosis leads to constant alcoholic stimulation, or the onset of physical or mental depression in an alcoholic precipitates the attack of polyneuritis.

Morbid Anatomy.—*Lesions in the Nerves.*—In the very great majority of cases the condition found in the nerve-trunk is one of degeneration comparable to that caused by traumatism, with the difference that in polyneuritis the nerves show many axis-cylinders in a comparatively normal condition. Indeed, all grades of Wallerian degeneration may be encountered in a single nerve, the fibers being differently affected by the toxic agent. It is also evident that the intensity of the toxic process and the duration of the disease will produce corresponding modifications in the nutrition of the axis-cylinder. It is possible that eventually we will be able to distinguish a variation in the degeneration produced by the various provocative agents, but at present they escape detection. The degeneration following lead-poisoning is apparently identical with that caused by toxins. The intensity of the neuritis generally decreases from the periphery toward the centers. Hyperemia of the endo- and perineurium is frequently observable. In addition to the axis-cylinder changes in some cases, usually those of long standing, there is an increase in the adventitia. A considerable thickening of the nerve-trunk results. This fibrous proliferation may even be excessive and at exposed points sometimes produces nodular enlargements. Syphilitic, leprous, and tubercular changes incident to these various diseases are also encountered. In the leprous form the nerve may also contain the characteristic bacilli. Ceni,[1] Crocq,[2] and others, by inoculation experiments in lower animals, have produced the same changes in the nerves and in the spinal cord as are found in human subjects, but not in the brain.

Lesions of the Spinal Cord.—In a considerable and constantly growing number of cases the cellular parts and even the conduction tracts of the spinal cord have shown involvement. Oertel, Dejerine, and Pernice have described lesions of the anterior horns in diphtheric neuritis. Many, among them Finlay, Achard, Schaeffer, Larkin and Jelliffe,[3] and Clarke,[4] have noted the same thing in alcoholic neuritis. Others again, as Thiersch and Rosenbach, recognize a simple atrophy of the cornual cells in lead-palsy. The cells of Clarke's column and the fibers of the posterior and cerebellar tracts have presented diffuse lesions of an inflammatory and degenerative nature. Henschen [5] records a case of typical diphtheric polyneuritis followed by acute disseminate sclerosis. The spinal cellular changes consist either of a changed crenelated form with vacuolation, or of changes of an inflammatory character, sometimes with hemorrhages. The lesions of the spinal white matter are principally degenerative.

Lesions of the Brain.—Pernice and Scagliosi [6] in diphtheric cases found the principal changes in the brain, cerebellum, and cord, and referred them to circulatory disturbance followed by degenerative changes and hemorrhage. The cortical cells showed atrophic degeneration, which, as in the cord, especially affected their protoplasmic prolongations. The well-known effects of alcohol and lead on the cerebrum are also found. Dehio,[7] experimenting on animals with poisonous doses of alcohol, found by appropriate stains that many of the cortical cells were changed in whole or in part. The presence of psychical symptoms in many cases of polyneuritis abundantly proves that the affection is one that does not spare the highest nervous apparatus.

On the other hand, in many cases of polyneuritis no changes have been detected. Thus Hosche,[8] in a carefully examined case of widespread diphtheric palsy, found absolutely no change, even in the muscles which had been paralyzed. He, therefore, attributes the symptoms to toxins which operated without causing anatomical changes. The *optic nerve,* which is, in fact, a cerebral structure, is often affected in polyneuritis. We have here to recall the whole list of toxic amblyopias. Alcohol again plays the most frequent part. The *medulla* and the *cranial-nerve nuclei* are subject to the same changes as the analogous portions of the spinal cord.

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[1] "Rif. Med.," Feb. 5, 1896.

[2] "Rev. Men. des Mal. de l'Enfance," Sept., 1895.

[3] "Med. Rec.," July 8, 1899.

[4] "Br. Med. Jour.," Sept. 12, 1903.

[5] "Fortschritte der Medicin," 1896.

[6] "Rif. Med.," Oct. 8, 1895.

[7] "Centralblatt f. Nervenh. u. Psych.," March, 1895.

[8] "Münch. med. Wochens.," March 12, 1895.

Lesions in the Muscles.—The muscles supplied by the affected nerves in polyneuritis undergo changes similar to those in simple neuritis or in nerve-division, but usually less in degree. The escape of numerous fibrils in the nerve-trunks is probably to be correlated with the persistence of numerous apparently normal fibers in the atrophied muscles. Another and important feature, however, is added, and that is the tendency to fibrous hyperplasia in the affected muscles, constituting in well-marked cases an *interstitial myositis*, which, in its turn, may act detrimentally upon the sarcode elements. This muscular fibrosis also explains in part some of the tendinous contracture deformities of late cases. The presumption is that an element of irritation is present which acts through the apparently normal nerve-fibrils, though Babinski is disposed to attribute it to the pathogenic effect of the toxic agent directly on the muscle.

Changes in the *trophic* and *secretory functions* of the nerve are, like the motor and sensory, less pronounced than in a simple neuritis, but do appear, and are marked by disappearance of the fatty panniculus, by thinned and glossy skin, vascular disturbances, mottling, edema, lack of perspiration, and by modified growth of hair, nails, and epithelium.

Symptoms.—The symptoms arising from so wide-spread a disease are correspondingly varied. It is necessary to take them up systematically, but it may be said, once for all, that they are symmetrical, as a rule, affecting both sides of the body in a similar manner. This might be expected from a toxic cause of systemic distribution. It is, however, a fact that the symptoms usually commence first on one side and frequently are slightly more marked on one side than on the other throughout the disease. They may be alternately exaggerated on the two sides. Some toxic agents, as lead, affect more especially the upper extremities; others, particularly alcohol, principally involve the lower. Again, in some cases the motor symptoms predominate; in others, the sensory.

Muscular and Motor Symptoms.—*The Lower Extremities.*—The motor disturbance in polyneuritis is principally one of deficit, a paresis that in severe cases reaches complete paralysis. It is most marked at the distal extremities of the limbs, and in the great majority of cases affects first and most the extensor muscles. In the lower extremity the extensors of the toes, the peroneal muscles, and the dorsal flexors of the foot are the ones usually most implicated. In marked cases foot-drop is complete. The foot falls into line with the leg and the plantar surface turns inward, passively producing an equinovarus position when the patient is recumbent, or when the foot is raised from the floor (Figs. 115 and 116). In milder cases the patient can not raise the toes from the floor while standing on the heels, or, in other words, can not dorsally flex the foot beyond a right angle. The toes remain in a flexed, bunched position, and can not be extended or separated. Efforts against resistance at once disclose the weakness of these muscles. A peculiar and *characteristic gait* is developed. In order to clear the ground the patient is compelled to raise the foot by flexing the thigh. The leg is then thrown forward like a flail; the toe is pendent; the outer border of the foot is depressed and brought to the ground toe first, or in a flat-footed manner, or the outer border of the foot strikes the floor first. The other foot is then advanced in precisely the same manner. From this high knee-action Charcot denominated the gait "steppage." The dangling foot sometimes wears the boot at the toe and frequently scrapes up the dirt of the pavement. In very rare instances a contrary position of the foot and an opposite sort of gait has been produced by loss of power in the calf-muscles, the anterior group being spared. The patient in such cases in walking brings the heel first to the ground with the foot everted and dorsally flexed.

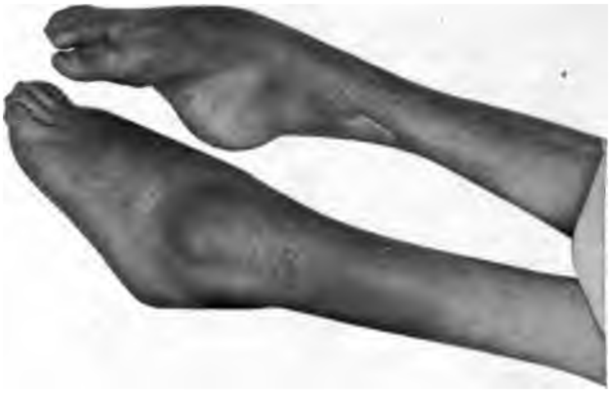

Fig. 115.—Drop-foot and deformities in alcoholic multiple neuritis.

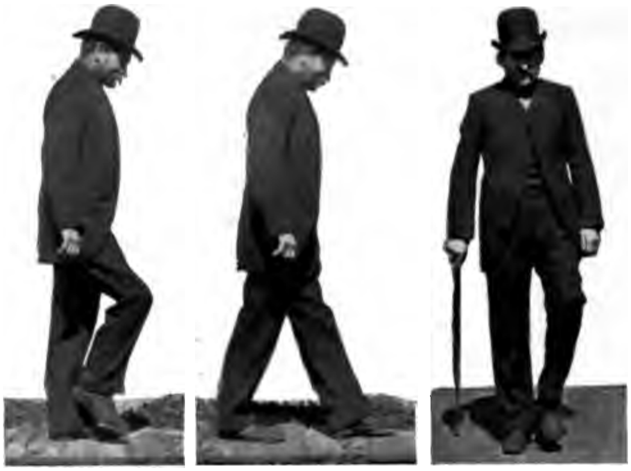

Fig 116.—Gait in multiple neuritis, showing the high knee-action and the dangling foot descending by its toe and outer border.

When the thigh-extensors are also affected and the muscles at the root of the limb are weakened, the use of the member becomes well-nigh impossible. A paresis of the extensors of the knee is often associated with that of the leg muscles in alcoholic polyneuritis. As above indicated, the loss of power may be trifling or absolute. While the muscle-groups enumerated are most affected, their opponents also lose power, but in less degree. The loss of synergy alone would account for much of their weakness, but the neuritic process does not entirely spare them, and in severe cases of long standing they also waste.

The Upper Extremities.—The distal segments of the upper extremities, as in the lower limbs, are principally involved. The muscles of the forearms and hands are most affected. In polyneuritis the muscles under the control of the musculospiral are usually the first to suffer, the most involved, and the last to recover. *Drop-wrist*, the attitude characteristic of musculospiral palsy (see p. 293), is, therefore, a prominent deformity, and the disability of the hand conforms to this type, but the supinator longus often escapes. In some instances, to the drop-wrist is added the deformity characteristic of a median-nerve palsy, and the claw-hand or ape-hand is developed. The postcarpal tumor may also appear. In still rarer cases the deltoid, biceps, long supinator, spinati, and short supinator first suffer, but ordinarily they are involved, if affected at all, after the forearm- and hand-muscles have been invaded. These functional groupings of the palsies of polyneuritis again furnish us with evidence of the essentially nuclear character of the lesion.

The trunk-muscles are sometimes also weakened, but only after the paresis of the extremities has become pronounced. Standing, walking, and even sitting may thereby be enfeebled or completely prevented.

The muscles of the neck and face in very severe and extensive cases may also be affected, as may the muscles of the *eyes*, *tongue*, *throat*, *larynx*, *diaphragm*, of respiration, and of the heart itself. Involvement of the muscles of the throat, palate, and accommodative visual apparatus is a characteristic combination in diphtheric palsy. Brunton [1] calls attention to a mask-like face, frequently seen in the alcoholic variety of multiple neuritis, affecting particularly the portion of the face between the eyes and lips.

Electrical Changes.—Owing, probably, to the variable amount of injury and irritation in the nerves, electrical tests in polyneuritis give all possible changes from the normal in different cases, and frequently at different periods in the same case. Not infrequently in the early days of the disease electrical excitability to faradic and galvanic currents is somewhat increased. This lasts but a short time, when diminished faradic response is presented, and may be attended in rare instances by voltaic diminution, a purely quantitative change. More frequently and customarily, as faradic excitability diminishes, the voltaic current augments in power and eventually presents some or all of the items of the reaction of degeneration (see p. 47). Complete abolition of electrical response to the galvanic current is, however, rare, even in the severest cases. Popoff [2] contends that diminution of electrical responses may be detected long before paresthesia is noticed. Erb has noticed that with the increased voltaic responses sometimes *myoidema* can be easily produced. A sharp blow across the muscles with the handle of a percussion hammer, especially near the tendinous portions, produces localized contraction of the muscle-fibers, causing a swelling that lasts for several minutes.

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[1] "Br. Med. Jour.," Dec. 1, 1900.

[2] "Neurol. Centralbl.," 1900.

Muscular atrophy follows the muscular enfeeblement, which in its turn is usually preceded by sensory disturbances. The muscular tone sinks as the paralytic features develop and amyotrophia is promptly added. In rare cases the loss of power is pronounced and yet the muscular masses retain their usual conformation and electrical responses, but gradually their swelling curves are replaced by straight lines or even by hollows, and the wasting in extreme cases apparently denudes the bones of everything but the dermal and fibrous structures. A polyneuritis may stop at any point or attain any degree of intensity. The muscular atrophy necessarily shows corresponding variations. As the paralytic features preponderate in the extremities and particularly in the distal portion of the limbs, the amyotrophia is most marked in the same positions. We, therefore, find it ordinarily in the hands and feet, in the forearm and below the knee, but there is no voluntary muscle that may not be involved, and even the internal viscera are not immune. In some cases a thick layer of subcutaneous fat or an edematous condition may disguise the wasting of the muscles.

Other Motor Disturbances.—The lack of muscular balance in some cases and the diminution of muscular strength, whenever pronounced, may be attended by a certain amount of coarse *trembling*, which is likely to appear on voluntary effort or when the muscles are strongly exerted. This, taken with the disturbed sensation, especially in the fingers, leads to much *clumsiness* in the smaller coördinate movements of the hand. Most patients early find difficulty in picking up small objects, buttoning their clothing, etc. For similar reasons they are not so precise in the use of the lower extremities, and from the loss of sensation in the feet they sway and sometimes fall in attempting to stand with closed eyes. The gait may also appear *ataxic*, but in the sitting or recumbent position these patients direct the movements of their lower extremities with as much exactness as their limited power will allow. The same is true for the movements of the upper limbs. The apparent ataxia must usually be attributed to muscular weakness. *Fibrillary twitching* in the atrophic muscles is frequently seen. Cramps sometimes occur upon effort being made after a period of repose, or spontaneously, or after the use of a comparatively mild faradic current. Patients very frequently complain of cramps, and of cramped positions. Hyperesthesia and other sensory disorders probably lead to such complaints.

Contractures are very prone to develop. Their disabling character can not be too strongly emphasized, the more so as the malposition to which they give rise can usually be prevented by proper, early management. They arise: (1) In part from the lack of balance in the muscles on the two sides of the limbs; (2) in part from the natural tendency of the flexors to impose upon the joints a position of demiflexion; (3) in part from the action of gravity, and (4) in great part from the development of the muscular fibrosis and fibrotendinous retraction in the muscles incident to the disease. From these causes the foot-drop becomes a fixture. Attempts to dorsally flex the ankle-joint are stopped abruptly by the rigid, shortened heel-tendon, which alone impedes the joint's movements. A similar condition is often present at the knee, elbow, wrist, and even at the hip and shoulder. In exceptional cases tenotomies are eventually required to enable the patient to put the heels to the floor.

The Reflexes.—The *cutaneous reflexes* in the parts affected by a polyneuritis may be normal, but are usually diminished in activity and sometimes abolished. The *tendon reflexes* are almost always diminished or abolished in the limbs that are most affected, but, of course, only in the muscles that suffer. The Achilles jerk may be abolished and the knee-jerk present, or there may be some difference on the two sides of the body. In well-marked cases, however, the ankle, knee, wrist, and elbow reflexes are usually greatly diminished and often wanting. Occasionally in slight cases, and then usually only at first, the reflexes may be very active, or even exaggerated, but clonus is never encountered. The *organic reflexes* governing the intestine and bladder are very rarely impaired and practically never abolished. Sphincteric control is, therefore, maintained.

Sensory Symptoms.—The sensory disturbances of polyneuritis are usually the first to appear, the last to cease, and they often, even more than the paralytic features, distress and worry the patient. Popoff,[1] however, contends that reduced faradic reactions are the earliest symptoms, and the author has foretold multiple neuritis in alcoholic cases by the diminished knee-jerks weeks before there was any complaint of paresthesia. Most cases begin by *paresthesia* in the extremities, which the patients describe by such words as tingling, numbness, dumbness, pins and needles, coldness, heat, crawling, tickling, and similar expressions. They shortly notice that tactile impressions produce abnormal sensations, and it often requires careful questioning and examination to determine whether the condition is one of hypersensitiveness, blunted sensitiveness, or both combined. A light stroke with a feather may give rise to a distinct sensation of tingling, when the patient can not tell whether he has a small object, such as a pin, between the tips of his fingers and thumb, or may not be able to recognize such a familiar object by the sense of touch. As a rule, these sensory disturbances are persistent when once established, and only change gradually for the better or worse. In extent they present the greatest differences, depending upon the activity of the neuritis and its duration. Commencing usually in the finger-tips and in the toes and soles of the feet, they gradually extend up the limbs, enveloping them as if they were submerged in some loose material like cotton or shavings. Commonly the disturbance does not go above the elbow and knees, but may reach the roots of the limbs and invade the trunk. In a number of cases similar paresthesias are felt in the face, lips, and tongue at an early stage of the malady.

As the disease advances, these persistent subjective sensory symptoms become aggravated and frequently marked by crises of intense suffering, which are usually of the same character in a given case, but vary widely in different patients. One will complain of the most intense pain; another suffers with burning sensations and wants cold applied; a third feels that the extremities are cold to freezing and in a warm room bundles them with woolens. As a rule, when pain or sensory suffering is pronounced it is most marked in the lower extremities. It often deprives the patient of sleep and is an exhausting element in the case.

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[1] "Neurol. Centralbl.," No. 13, 1900.

Sensibility to touch, pressure, painful impressions, and temperature may also undergo marked changes, which are infinitely varied. The cutaneous sensations are disturbed in areas roughly corresponding to the paresthetic distribution and the motor loss. Early in these cases, when the irritative conditions in the nerve are probably at their maximum, there is a tendency to *hyperesthesia*. The patients may complain that their stockings feel as though full of knots, that the bedding feels as if made of ropes, and in various phrases express the exaggerated modifications of touch. Light contact may be painful. Pressure on the nerve-trunks and on the muscular masses usually develops marked *tenderness*, and in severe cases may evoke a painful crisis. In many marked cases some dissociation of cutaneous sensations is observed. The pain and temperature sensations may be diminished more than tactile impressions, or touch may be abolished, the contact giving rise to pain or discomfort, which may not be properly located. In cases where the local conditions of wasting, anesthesia, and electrical tests indicate marked nerve-degeneration, the patient may still complain of pains, burning, etc. In late cases *anesthetic features* are nearly always present, but it is rare for them to reach a complete degree. Some modifications of the muscular and joint sense are also encountered, so that the blindfolded patient may have uncertain knowledge of the position of the limbs. The transmission of all forms of cutaneous sensation may be retarded, and generally all forms are uniformly decreased.

Various toxic substances differ in the sensory disturbances they produce. Some, with alcohol and grip at the head, produce marked painful disorders of sensation; while others, of which lead and diphtheria are the chief, mainly cause motor impairment,—another argument for the central location of the disturbing element.

Ocular Symptoms.—*Vision.*—Among the special senses, that of vision is the one principally, if not alone, affected. Many of the substances capable of producing a polyneuritis also engender a toxic amblyopia. These are especially alcohol, lead, arsenic, and the carbon gases. (For the special symptoms which pertain to them, reference should be made to p. 103, and to special works.) It must be borne in mind that even a pronounced optic papillitis, retracted fields, color scotomata, and considerably reduced vision may fail to attract a patient's attention and must be intelligently sought. In the toxic cases the condition is bilateral and symmetrical, and usually of insidious development.

Motion.—Squints and diplopia have been noted as frequently as once in ten times in diphtheric neuritis (Remak) and occur also, but less frequently, in the alcoholic form. They are due usually to a neuritis of the oculomotor nerves, but sometimes to nuclear involvement, when they are commonly attended with apoplectic features and are of sudden onset. The motor nerves of the eyeball may be affected equally or singly, producing corresponding deviations, ptosis, etc. Commonly single muscles are paralyzed, a complete ophthalmoplegia being usually of nuclear origin.

The Pupils.—In rare cases of polyneuritis the pupils have been affected. Dilatation, contraction, and inequality have been noted by various observers. The pupillary reflexes, however, are unimpaired, and the Argyll-Robertson phenomenon is not seen. Brunton contends that the pupillary response for accommodation is lost, that for light remaining active, the converse of the Argyll-Robertson phenomenon.

Accommodation is often disturbed in diphtheric neuritis, of which it is one of the most constant symptoms. Here, too, the pupils remain active. As a result of the paralysis of accommodation vision for near objects is reduced. This can be detected even in very young children by asking them to note a pin-hole in a calling card. For older patients printed characters are blurred and illegible.

Trophic and General Conditions.—In addition to the muscular atrophy, the trophic disturbance is manifest in the extremities, especially toward the digits. The skin is ill-nourished, cold, muddy in complexion, often turgid and reddened. It is often glossy about the nails and the fingers are tapered with overhanging, claw-like nails, which the patient sometimes refuses to trim owing to the tenderness and hyperesthesia. Edema is not infrequent and rarely an extra deposit of fat is encountered, as also in the condition named adiposis dolorosa by Dercum, but ordinarily the fat disappears early. Perspiration is reduced, or more rarely increased, causing a sodden crumbling of the epidermis. The nails and hairs may become coarse, stiff, and fragile. Herpetic eruptions, erythema, and ulcerations, so frequent in an ordinary neuritis, are very uncommon.

The general nutrition is usually greatly reduced and slight elevations of temperature are common. In the infectious forms fever may naturally be expected. The prolonged toxic or cachectic state, which usually antedates the onset of the polyneuritis, accounts for much of the physical depression, but some of it is apparently due, and its continuance is partly attributable, to the lowered functional activity that pervades the entire nervous system. In severe and prolonged cases difficulty in swallowing, an inactive stomach and intestine, a weakened heart and failing circulation, the continuance of pain, with perhaps insomnia and mental disturbance, produce a marantic depression that ends fatally or destroys resistive power against acute infections such as pneumonia. But usually the general state can be maintained at a satisfactory level.

Mental Symptoms.—Following Korsakoff, a number of writers have sought to delimit a psychosis peculiar to polyneuritis. It was pointed out by Colella[1] that such mental disturbance occurred only when the patient was hereditarily neuropathic. Among others, Ferrari [2] has shown rather conclusively that this so-called polyneuritic psychosis is merely the mental state that marks chronic poisoning by alcohol, lead, and other toxic substances, and has something in common with the confusional insanity that sometimes follows infectious fevers. Charcot maintained that it was related solely to alcohol, but Bury [1] has noted it in the Manchester epidemic due to arsenic in very moderate beer drinkers. Even here, however, an alcoholic influence can not be denied. However, the association of mental disturbance with polyneuritis is extremely common and in slight degrees is easily overlooked. It is most often encountered in the alcoholic variety, then in lead cases and the infectious multiple neuritides. In some cases the mental condition precedes the appearance of neuritic phenomena, but more commonly follows the onset of the polyneuritis and develops progressively with it. It is first manifest in a change of temperament and character. The patient becomes irritable, capricious, and exacting, or may show apathy and indolence. This is attended or followed by loss of sleep and the nights are often marked by agitation and vague alarm, which subside with the day, to return at evening. Hallucinations develop as the malady advances and may lead, in the depressed and apprehensive mental state, to impulsive acts of a self-protective or defensive character. The patient may thus attack or abuse those about him or seek safety in flight. These marked mental symptoms may still subside toward morning, but finally in some cases the delirium persists night and day. When this cerebral condition occurs it usually appears comparatively early in the case, and corresponds, perhaps, to those irritative features that have been indicated in the peripheral nervous apparatus. Convulsions sometimes most clearly and emphatically demonstrate the cortical irritation.

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[1] "Rivista Sperimentale," vol. xx.

[2] "Alien. and Neurol.," Jan., 1896.

As the paralytic features develop in the limbs, a partially demented condition obtains for the cerebrum. Often the period of *delirium* and excitement is not evolved, and the depressed mental condition is insidiously developed primarily. This is marked most prominently by a *forgetfulness* which pertains to the immediate past experiences and does not notably disturb the patient's recollection of events and conditions prior to the illness. Such a patient may talk rationally and logically on every possible subject, but is likely to forget what he has said or heard only a few moments previously, and repeats remarks and questions at short intervals or tells the same story over and over. Because of this amnesia he is frequently at fault as to the day of the week or month, or even the hour of the day, and may be unable to tell at noon what he had for breakfast. This is in most striking contrast with the patient's ability to remember the slightest details of his early life, with his apparent consciousness of his surroundings, and with his ability to logically associate facts presented to him.

In addition one often observes what at first would seem to be the play of a diseased imagination. These patients may assert, perhaps when already confined to bed for months, that they are just returned from a long journey, of which they give endless and minute details, name persons encountered, repeat conversations held and business transacted, when they were, in fact, unable to turn over in bed. Such tales are recounted in the most ordinary way, without the slightest excitement, and completely forgotten in a short time. If we analyze them we find a mass of more or less definite incidents or purposes transferred from the patient's past life and brought down to the date at which they are recounted, the intervening time having disappeared. Patients will speak of having just seen relatives long dead, or give directions for the entertainment of persons who may be in distant parts of the world. If their attention is called to these discrepancies they first try to maintain their statements, but readily become mentally confused and uncertain.

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[1] *Loc. cit.*

This amnesic mental condition may be developed in all grades of intensity, and may even proceed to complete dementia and a fatal termination. The bodily strength and powers of assimilation usually are correspondingly depraved. The mental disorder passes away as the case mends, and the mind is usually perfectly clear before the peripheral apparatus is fully restored.

Course and Special Forms.—Polyneuritic cases present many clinical varieties and some special forms. An ordinary case may be outlined as follows: The first complaint is of tingling or formication in the feet and legs, with occasional pains. After a few weeks the hands and forearms are similarly affected and some slight loss of muscular strength is recognized. The sensory and motor troubles increase. Pain becomes more prominent and the paralytic features invade the thighs, hips, and perhaps the trunk and arms. After about eight weeks the malady is full-fledged. The patient now walks with the high action, the muscles of the extremities are notably wasted and give the electrical reaction of degeneration, foot-drop and wrist-drop are well marked, and the reflexes are diminished or abolished. The patient is uncertain on his feet, the legs and feet readily become edematous, red, and livid ; with closed eyes he sways and perhaps falls. In bed, from the weakness in the arms and trunk, he can not gain the sitting posture without help. The muscles and nerve-trunks are sensitive to pressure ; there is some anesthesia or at least much blunting and retardation of the cutaneous sensations, and much sensitiveness in certain cases. The skin is dry and thinned about the digits and base of the nails. At this time mental disturbance is likely to appear. After this state is reached the case remains almost stationary for several months; then the sensory disturbances diminish slowly, the pains decrease, the anesthesia disappears; the muscles regain their former proportions, strength, and reactions; the reflexes reappear, and full recovery is established, after about twelve months from the onset of the disease. Very often anesthetic areas become hyperesthetic and the pains increase as regeneration passes downward. In some instances the disease, instead of coming to a standstill, progresses to a fatality by involving the muscles of the trunk, of respiration, deglutition, and circulation. Death results from pneumogastric involvement. In other cases the development of fibrotendinous contractures disables the patient after convalescence until surgically relieved. Again, the progress of the disease may be much varied. It may commence insidiously and then advance with great rapidity, or, commencing suddenly, quickly reach its maximum and then slowly or quickly subside. When atrophy is well marked, restoration is always a tedious affair. As a rule, in the cases that recover full health is regained, but in a few cases localized atrophies and contractures remain permanently.

Other clinical varieties depend on the location of the prominent symptoms of the disease. The cranial nerves, the upper extremities, or the lower limbs may be most affected. Some cases show a preponderance of motor disability, others of sensory disturbance. The rare cases marked at first by increased reflexes, increased electrical responses, marked hyperesthesia, and perhaps a tendency to delirium, must not be overlooked.

In addition there is a small group of very acute cases in which the disease, beginning in the feet and legs, rapidly invades the thighs, the trunk, the upper extremities (commencing in the fingers and gradually passing up the limbs to the shoulder), and, finally involving the diaphragm and the bulbar nerves, reaches a fatal termination in from six to twenty days. By some writers this form is considered identical with Landry's paralysis, but by others is distinguished from it by the disturbance of sensation and reflexes and the postmortem presence of changes in the nerve-trunks. Any sharp distinction is arbitrary and untenable, as all gradations between multiple neuritis and acute ascending myelitis are encountered.[1]

Certain toxic elements cause a polyneuritis marked by peculiar and even characteristic symptom groups that merit brief attention.

The alcoholic form, besides being the most usual polyneuritis, presents with considerable uniformity the following features: The lower extremities are the most affected, and the painful sensory disturbances reach their highest range in this toxic state. They are proportionately much more pronounced than the paralytic conditions. The paresthesiæ are particularly intense and intolerable. The muscles and nerves are especially sensitive, and cutaneous hyperesthesia is exquisitely developed. The high-action gait is usually well marked, as the extensors of the leg are generally affected with the muscles below the knee. Atrophy is well marked and the tendency to fibrotendinous contracture is very strong. Involvement of the ocular muscles is comparatively frequent and bilateral scotomatous amaurosis is common. Of all forms, alcoholic polyneuritis furnishes by far the greatest number of cases of mental disturbance. Recovery is comparatively rapid if alcohol can be withdrawn. Relapses are probable, and a single indulgence before the disease is definitely at an end may cause the most startling reappearance and exaggeration of all the former symptoms.

Lead-palsy in typical cases presents some or all of the following conditions: It usually comes on after prolonged exposure to intoxication through the patient's occupation, or by the use of drugs and cosmetics, or by drinking contaminated water. The source of the lead-poisoning is often extremely puzzling and requires the most painstaking search. The paralytic features are usually preceded by colics and constipation, and may develop at a long period of months after the patient has been removed from the source of intoxication. In other cases the administration of alkaline iodids to one impregnated with lead, but showing no marked symptoms of it, may precipitate the neuritis. The upper extremities are first and almost exclusively affected. The distribution of the palsy is practically symmetrical, but the right hand is most disabled in right-handed patients, the opposite hand in the left-handed. Sensory disturbance in the hands is practically absent, but if the legs are affected cutaneous sensibility is changed and pains are usually present below the knees. The muscular wasting is prominent, and in some cases seems to be the primal disorder, bringing these cases into apparent relation with the myopathies. Most cases show the wrist-drop feature, but arm and shoulder types may be encountered alone or may be gradually added to the first form, establishing an appearance of relationship with the spinal myopathies. The muscles of the larynx are frequently affected, as shown by aphonia. The retrocarpal tumor is usually developed. Ocular disturbances are frequent, and due to optic neuritis, from which sudden blindness may occur. When the trunk and bulbar muscles are involved, the outlook is better than in the alcoholic variety under similar circumstances. The course is usually insidious at the beginning and protracted throughout.

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[1] Medin, "Arch. de Med. des Enfants," 1898.

Lead-palsy also has its associated mental disturbance. In rare cases this resembles the one so usual in alcoholic polyneuritis sketched at page 318, with marked visual hallucinations and amnesia. Lead, however, has its own cerebral disorders. These are an acute mania and a condition that presents all the somatic indications of general paresis, but is usually marked by mental sluggishness and is devoid of the expansiveness of true paretic dementia. In addition lead may engender general cerebral affections by its action on the circulation, mainly by the arteriosclerosis and kidney disorders it induces. Delirium, convulsions, coma, hemiplegia, and combinations of these are frequently encountered in plumbism.

Diphtheric Paralysis.—The term paralysis is selected purposely, as a neuritis can not always be demonstrated in these cases. Hochhaus found only the muscles affected, and Babinski, in rabbits paralyzed by injections of the toxins derived from the Klebs-Löffler bacillus, found no changes whatever. In certain cases, however, a periaxial neuritis is clearly demonstrable. What the conditions are that determine a neuritis in one case and leave no trace in others can not now be stated. The result in both instances is undoubtedly due to the action of the toxins evolved in the life-history of the bacillus of diphtheria. Diphtheric palsy may appear after all forms of the initial malady, whatever its apparent intensity or bodily location. Ware, for instance, reports a typical case with the usual pharyngeal palsy, the diphtheric membrane being located on the vulva. It seems to be rather more common in adult cases, perhaps because the palatal, pharyngeal, and pneumogastric involvement in infants may lead to a fatal termination without being attributed to a nerve-lesion. Ordinarily, it appears during convalescence in from one to three weeks after the diphtheria has subsided, but may develop within a day or two of the appearance of the pseudomembrane. The muscles of the soft palate and the pharynx are the first to suffer, as a rule, and the paralysis may extend to the lips, cheeks, and tongue. If it spreads further, the legs next suffer. The knee-jerks are often abolished, even before the palate is disturbed. The upper extremities and the trunk are invaded only in exceptional cases. The muscles of the neck, on the contrary, are frequently weakened, so that thé head may roll about helplessly on the shoulders. The affected muscles rarely waste and contractures hardly ever develop. Sensory changes consist of insensibility in the affected parts, as in the pharynx, larynx, etc. Much pain and painful paresthesia are wanting. There is usually some ataxia in the extremities and Romberg's sign is seen. At the time the palatal difficulties appear there is usually some loss of visual acuity, especially for near objects, which is due to the characteristic paralysis of accommodation, but the pupils do not fail to act. Strabismus, double vision, and ptosis are not uncommon. In adults, sexual impotence is frequently developed. Mental symptoms almost never occur. Diphtheric palsy is of rapid development and usually terminates in recovery. If only the palate is affected, cure is reached in two or three weeks; in more generalized forms the disability is prolonged to three or four months, or even to a year in severe cases. If death occurs, it may be attributable to the cachexia and inanition from inability to swallow or may be caused by pneumogastric paralysis. Inspiration pneumonia is also likely to carry off the patient. In very rare cases of diphtheric palsy the paretic features are fleeting, and change from place to place or disappear and return. These cases are more frequent since the general use of antitoxin in the treatment of diphtheria, probably because cases are now saved that otherwise would have terminated fatally.

Erythromelalgia is a variety of multiple neuritis affecting principally the plantar nerves, and first described by Weir Mitchell as a vasomotor neurosis. Collier [1] reports ten cases of erythromelalgia in organic nervous disorders: six of insular sclerosis, three of tabes, and one of myelitis. It may also occur with decided arterial changes, but undoubtedly is a neuritis, as proven by Mitchell and Spiller.[2] It usually attacks men, and is worse at night and after walking. The feet become intensely red and show some swelling. The pain is of a burning sort and the attending perspiration may be profuse. Heat aggravates and cold relieves the distress. An elevated position also affords relief. Walking may become so painful as to be impossible. Trophic disturbance in the skin in severe cases is encountered. The condition may get well spontaneously or be very persistent. Rest in bed, electricity, massage, and, in protracted cases, excision of the posterior tibial nerve have been advised.

Beri-beri, or kakké, is a polyneuritis that apparently depends upon a specific infection. Several investigators have found various micro-organisms which they have thought to be the pathogenic agents, but there is as yet a lack of uniformity and confirmation. There are those who insist that it is due to carbonic gas poisoning. Others have attributed it to a diet of fish or rice. It arises when people are huddled together in prisons, asylums, barracks, or on shipboard, and is particularly common in China, Japan, and India, and in South and Central America. It is marked by paralytic and atrophic disorders, especially in the lower limbs, and particularly in the antero-external leg muscles, producing the characteristic gait. The phrenic and pneumogastric nerves suffer early, and respiratory and cardiac symptoms are prominent. The face and tongue are frequently affected. The sensory disorders are mainly anesthesia and severe lightning-like pains. Edema of the lower extremities is often very great. It may also invade the trunk and flood its cavities. The knee-jerks are lost early. There is frequently right cardiac dilatation, rapid heart action, and murmurs over the base. Mental disturbance, similar to that in alcoholic cases, is frequently observed. All degrees of severity, from simple weakening of the lower extremities, with cardiac palpitation, to a pernicious acute form, like an acute ascending myelitis that runs to a fatal termination in a few days are presented. Intermediate varieties may be marked most by atrophy or by edema, and are very long in recovering, with death always likely from pneumogastric accidents. A large proportion of cases are said to have intestinal parasites. H. Wright[1] found changes in the cells of the posterior root ganglia and the anterior horns of the cord in eight consecutive cases.

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[1] "Lancet," Aug. 13, 1898.

[2] "Am. Jour. Med. Sci.," Jan., 1898.

Leprous Neuritis.—The invasion of the nerves by the leprous bacillus sets up a multiple neuritis. There is a marked tendency to fibroid proliferation in the nerve-trunks, which often become nodular, and eventually the bacilli disappear. The spinal cord is exceptionally invaded by the bacillus (Souza, Martius), and presents central cavities, especially in the posterior horns and gray commissure. The prominent sensory disturbance is anesthesia, which occurs in discrete plaques, favoring in location the hands, feet, forearms, legs, and face. By spreading, an entire member or a large portion of the body may become anesthetic, and the deep parts, muscles, bones, etc., are also involved. Muscular atrophies are comparatively slight, and most affect the small muscles of the hands, feet, and face. Atrophic conditions in the hands and feet lead to mutilations of the extremities. Fingers, toes, and even hands and feet necrose and are cast off. The evolution of the neuritis is extremely slow, and may extend over a score of years.

In some cases a dissociation of cutaneous sensation is encountered similar to that of syringomyelia, especially of the Morvan type, and probably due to the cord lesions previously noted.

Recurrent multiple neuritis as a variety has been described by Sherwood, Ross, Dreschfeld, Targlowa, Klumpke, and Osler. Thomas[2] also reports a case. It appears that certain individuals are susceptible to repeated attacks from various poisons, but particularly from lead and alcohol. It is not requisite that the patient be exposed to the intoxicant to induce a recurrence of the neuritis, and it is undetermined whether there is an original susceptibility or the first attack leaves a predisposition to its return.

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[1] "Br. Med. Jour.," June 29, 1901. [2] "Phila. Med. Jour.," May 14, 1898.

Diagnosis.—The diagnosis of a case of well-marked multiple neuritis, based upon a fairly full history and a detailed examination, can hardly be missed; but in the early stages, when a diagnosis is of most importance, it is frequently extremely difficult. The major items upon which it then rests are the motor and sensory symptoms, their symmetrical distribution, their predominance in the ends of the extremities, the more marked affection of the extensors, the modification of the reflexes, the tenderness of nerve-trunks and muscles, and the history or presence of some toxic agent capable of producing the neuritis. A multiple neuritis may be confounded with several diseases of the spinal cord, especially poliomyelitis anterior and locomotor ataxia. The following differential tables may help to distinguish them:

POLIOMYELITIS.	MULTIPLE NEURITIS.
Most frequent in children.	In adults.
Onset abrupt.	Insidious.
Embraces entire limbs.	Begins in ends of limbs.
Not usually symmetrical.	Symmetrical.
Immediate tendency to improvement.	Gradual extension.
Sensory symptoms slight or absent.	Sensory disturbance early and persistent.
No mental symptoms.	Mental symptoms common.
Usually leaves some deformity.	Recovers completely.

LOCOMOTOR ATAXIA.	MULTIPLE NEURITIS.
Girdle pains and lightning pains early.	No girdle pains; lightning pains infrequent.
Nerve-trunks often insensitive.	Usually oversensitive and often thickened.
Muscular sense disturbed early.	Only slightly disturbed or intact.
Amyotrophia and reaction of degeneration absent.	Develop early.
Peculiarity of gait due to incoördination and irrespective of muscular strength.	Due to paresis and proportionate to loss of power.
Strikes heels first and does not follow straight line.	Strikes toes and outer border of foot first and walks in straight line.
Circulation and trophic condition of limbs normal.	Muscular atrophy, edema, lividity, and epithelial changes.
Perforating ulcers, joint lesions, and osteopathies are common.	Rare or unknown.
Argyll-Robertson phenomenon usual.	Never present.
Optic atrophy common.	Rare, but toxic amaurosis frequent.
Vesical troubles frequent and early.	Very exceptional and late.
Gastric and intestinal crises.	Dyspepsias from toxic causes, constipation from lead, etc.
Fecal incontinence common.	Only in acute pernicious cases and in stuporous states.
Sometimes followed by paretic dementia.	Often accompanied by mental disturbance.
Of slow evolution, requiring years.	Of insidious, progressive development, requiring months.
Incurable.	Recovers if patient survives.
Syphilis usually in the history.	Antecedent intoxications, infections, and cachexias.

Myelitis is distinguished from polyneuritis by the girdle pains, the paraplegia, the anesthesias, the retention of electrical responses in many of the paralyzed muscles, and the presence of the deep reflexes, which usually are extremely exaggerated. In this disease bedsores are common; in polyneuritis well-nigh unknown. The sphincters are usually beyond ordinary control in myelitis and practically unaffected in neuritis.

Hysteria is sometimes confounded with polyneuritis, and, indeed, both may be present in the same patient, but should none the less be separately recognized. The pain in hysteria lacks sincerity of facial expression and the deep muscular masses are practically never sensitive. Hysterical anesthesia is segmental, and rarely involves all four extremities symmetrically. The reflexes and electrical reactions are present in hysteria—there is no wasting, no cutaneous dystrophy. The evolution of hysterical trouble, its emotional concomitants, and its stigmata are sufficiently significant.

The pernicious anemias often present reduced reflexes, paresthesias in the extremities, uncertain gait, mental vagueness, and general muscular weakness. A careful blood examination shows characteristic cellular changes (see page 452).

The diagnosis of the toxic cause and its origin is usually made in securing the history or in making the examination of the patient, but many times presents great and even insuperable difficulties. The alcoholic, lead, and diphtheric types of polyneuritis are themselves significant of their origin. Alcoholic addiction is frequently denied, especially by women. Patients may even be unaware that they are taking large quantities of alcohol in the form of various nostrums and patent medicines. The absence of the lead line on the gums should not mislead, as it only occurs where there is some local disturbance about the necks of the teeth. A dose of iodid, followed by an analysis of the urine, may show plenty of lead. Hair-dyes, face-powders, and styptic applications or injections may contain lead, and the polyneuritis may appear several months after their discontinuance. Arsenic and lead in fabrics, paints, wall-paper, toys, and medicinal prescriptions are to be borne in mind. In diphtheria, if the patient has been exposed, it is not necessary to have a pseudomembrane or even a history of a sore throat in order to attribute the palsy to the proper source. Leprosy and beriberi are distinguished by their infectious and clinical peculiarities. There remains the great class of infections arising from intestinal fermentation and the infectious diseases, which must be deciphered from the history and examination of the patient.

Prognosis.—It may be laid down as a general rule that if the cause of a polyneuritis can be removed, the patient has a very good chance of complete recovery, and that recovery is the most common termination of the disease. The prognosis is to be modified in accordance with the nature of the pathogenic agent, the mode of evolution of the disease, its intensity, extent, and localization, and the general physical condition of the patient. *Diphtherial* polyneuritis, unless the patient succumbs in the early days of the attack, is the most benign form, and rapidly terminates in recovery. *Lead* palsy alone very rarely causes death, but is usually attended by a bad general state and is of long duration and a disabling character. *Alcoholic* neuritis is more frequently fatal and is customarily associated with other organic disease due to the same cause.

The intense painful conditions that attend it and the debilitating suffering, with the tendency to mental disturbance, give to the alcoholic form a high grade of gravity. *Auto-intoxication* arising from the intestine usually is the result of chronic and intractable bowel disease, and is correspondingly protracted and unmanageable. *Acute invasion* and rapid evolution of a polyneuritis lead at once to the apprehension of respiratory and cardiac failure and an early fatal termination. On the other hand, an *insidious onset* and slow advancement imply chronic tenacity and a prolonged impairment of powers, with slow convalescence.

The gravity of a polyneuritis is in some proportion to its *extent*, particularly in relation to its advancement toward the trunk, implying great helplessness, and toward the pneumogastric, implying fatal accidents from its implication. The presence of *cerebral symptoms*, such as psychoses or convulsions, and of *spinal involvement*, as shown by loss of sphincter control, add greatly to the gravity of the outlook. The general state, the powers of digestion, assimilation, and the ability to sleep have a strong bearing on the recuperative prospects of the patient.

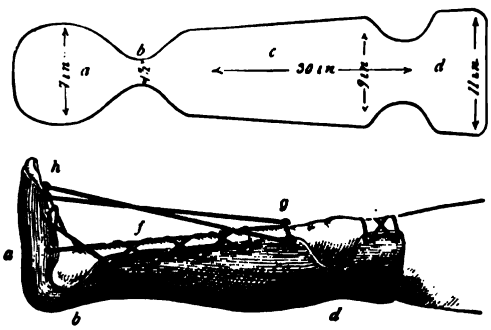

Fig. 117.—The upper figure represents the shape of the brown-paper pattern, and of the leather cut in correspondence to it. The dimensions are approximate, varying with each leg; *d* is the narrowing for the knee, that the leather may not press on the bone at the sides; *b* is the excavation on each side for the ankle with the narrow connecting piece of leather, which is the special feature of the contrivance. This, 1 to 1½ inches wide, and no longer than is needful, affords lengthwise, adequate support to make the pull from above act down on the foot-piece on which the pull acts; at *h* and *g* are the rings for the cords; the cords are here shown only as going to the lower rings; if there is contraction of the knee, they should be carried through these rings and fastened to the other rings above the knee; if there is no contraction of the knee, it is not necessary for the leather to extend above the knee (after Gowers).

Treatment.—The first element in the treatment of a multiple neuritis naturally is the removal of the cause. In alcoholic cases this is frequently a matter of great difficulty unless the patient can be placed under the care of a trained nurse or in a hospital. In some instances the immediate withdrawal of alcohol is attended with a great deal of physical and mental disturbance, but half-way measures usually only prolong the agony and frequently fail completely. After such a case has made decided improvement and if taken early this may promptly appear; a very little indulgence in alcohol is likely to cause the return of all former symptoms. Very moderate drinking is capable of maintaining them indefinitely.

Lead can be eliminated from the system by the use of the alkaline iodids and sulphates, but some care must be taken not to liberate the metal too rapidly, as cerebral disturbance and an increase of the paralytic features may be precipitated. They should be commenced in small but increasing doses, and aided by elimination from the skin, kidneys, and especially from the constipated bowels. Occupations giving rise to such poisoning must be given up entirely, as even when convalescence has been long established a relapse is likely to quickly follow a return to the former work.

In auto-intoxications from intestinal fermentation a restricted diet and the use of those antiseptics which tend to inhibit bacterial activity and reduce toxicity are indicated. In diphtheria the source of infection subsides with the cessation of the bacillary invasion. In the poisonings following acute infections the toxemic state requires attention and is best controlled by very large doses of iron, especially the tincture of the chlorid, by salol, and similar drugs. If a malarial element is present, quinin and arsenic are required. The various cachexias that are attended by the development of multiple neuritis unfortunately are rarely amenable to treatment. In tuberculosis and cancer, the measures which produce improvement in the general state benefit the neuritis.

The *general physical condition* in all cases requires attention from first to last, and one of the chief items in this connection is rest for the weakened muscles. This frequently means rest in bed. In all marked cases such rest is obligatory. In the acute cases that rapidly involve respiration and threaten the pneumogastric the greatest care must be taken to prevent pneumonic inflammation. At the same time the alimentation and support of the patient's strength frequently require the use of the esophageal tube by the way of the nose or mouth, or rectal feeding may be employed. From first to last, also, measures must be taken, especially in alcoholic cases, to prevent contractures and posture deformities. These usually are confined to the lower extremities. Foot-drop and inversion of the sole constitute the ordinary deformity, but the knees and hips are also frequently involved in a semiflexion made rigid by tendinous contractures and rarely by joint-adhesions. The weight of bed-clothing serves to increase the foot-drop, and must be taken off the toes. So far as possible the feet should be kept at a right angle with the legs and the knees and hips extended. The sensitiveness of the skin and the pains in the limbs often defeat attempts to use any fixation apparatus such as light splints, but if commenced early, they can often be worn with great advantage. Pillows may be so placed as to support the feet and remove the weight of bed-covering. Passive movements in full extension and especially full dorsal flexion of the feet on the legs should be gently used several times daily.

In early stages the use of hot or cold applications has been recommended, but probably does little good, aside from slightly relieving the pain. Vesication and strong stimulation by counterirritation must be avoided, owing to the likelihood of producing indolent ulceration. Vigorous massage and electrization that produces firm contraction of the muscles are also contraindicated at first. Gentle strokings or kneadings of the muscles that do not cause pain or distress are often very grateful to the patient, aid return circulation, and favor local nutrition. Hot baths, needle douches, Turkish and Russian baths, and vigorous measures generally should be reserved until the disease has passed its maximum.

The question of anodynes here, as in sciatica and other prolonged painful affections, is one requiring mature judgment and great conservatism. The coal-tar derivatives sometimes act well and are not so objectionable as opiates and cocain, which alone seem capable of controlling the pains in the severest cases. Chloral and bromids combined are the surest sleep-producers.

After the disease has reached its stationary period more vigorous local measures should be instituted. Massage of the weakened muscles and their exercise by galvanic electricity should be faithfully and systematically carried out once or twice daily. The wasted muscles frequently respond to static sparks before they will to the constant current, and this form of electricity, if available, will then render good service. A faradic brush or static sparks also seem to beneficially influence cutaneous anesthesia and hyperesthesia. The patient must be urged to use the limbs, and at the first possible moment to bear weight on the feet and get the heels to the floor.

In cases of long standing, when the contractures are strongly developed, stretching, under anesthesia, and the application of fixative dressings may be done, and, if this does not suffice, tenotomies are in order. In chronic cases of wrist-drop the use of splints, as described on page 293, will often be found of signal service. Here, too, courses of hot baths, especially if combined with intelligent massage, hot douches, the Scotch douche, and Turkish baths, are serviceable if the general condition of the patient does not contraindicate their use.

CHAPTER V.

HERPES ZOSTER.

HERPES ZOSTER, also known as *zoster*, *zona*, and more commonly as *shingles* (Lat. cingulum), is an acute infectious disorder beginning with pain, soreness and burning in a given segmental skin area, followed by discrete groups of papules on a reddened and swollen base, after a few days showing turbid and purulent contents, and later brown crusts which fall by the end of the second week, leaving reddened or pigmental patches and occasionally scars. There is sometimes fever in the initial stage which with the pain subsides upon the appearance of the eruption. Secondary infection of the eruption may complicate the course and termination of the disease. The *lesion* is an acute hemorrhagic inflammation of the corresponding posterior root ganglia. The favorite location is on the head or face and about the trunk, but any portion of the cutaneous expanse may be affected.

Etiology.—The exact nature of the infection in herpes zoster is not yet determined, but it presents seasonal, epidemic, contagious and self-limited peculiarities. To this infection, or the toxin therefrom arising, the posterior root ganglia show a decided susceptibility, but commonly one only is affected in a given case. Certain ganglia, moreover, are more prone to attack than others, namely, the Gasserian and those from the third dorsal to the second lumbar spinal roots. These are in relation with the gastro-intestinal tract by sympathetic afferent fibers. A similar eruption appears if the ganglion be secondarily involved in any inflammatory or destructive process, and injury to a nerve-trunk may also cause a herpetic eruption limited to its cutaneous distribution. Zoster sometimes occurs in connection with malaria,[1] typhoid, meningitis, and pneumonia, and after arsenical, carbonic oxid gas, and intestinal poisonings. Second attacks are so rare as to lend support to the idea that immunity is afforded by the first invasion. Herpes zoster is also frequent in tabes and paretic dementia, both of which commonly involve root ganglia.

Pathology.—Barensprung first found that zoster was associated with inflammatory changes in the nerve and ganglia. Head and Campbell[2] have placed the matter on a secure foundation. In all well-examined cases an acute inflammatory condition is found in the ganglion with bloody extravasation, destruction of cells and fibers, and inflammation of the sheath. These are followed by acute degeneration of greater or less amount and even by secondary sclerosis. In the *peripheral nerves* degeneration appears and disappears or may terminate in sclerosis pari passu with the change in the ganglion. The nerve changes are secondary to the ganglionic invasion, but an active hemorrhagic inflammation may extend into the nerve from the ganglion. The *spinal cord* presents acute degeneration of the root fibers in the posterior column, appearing about the tenth day of the disease. The vesicles of the skin eruption contain a sterile serum, and no evidence of bacterial invasion is found in the neighboring profoundly inflamed lymphatics.

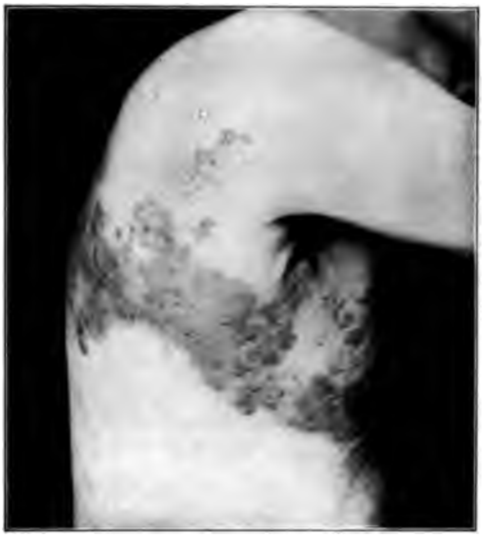

Fig. 118.—Herpes zoster confined to the fifth and sixth dorsal cord segments.

The disease may be considered as analogous to poliomyelitis anterior acuta, and the authors quoted have proposed for it the name acute posterior poliomyelitis.

Symptoms.—Zoster furnishes, according to Max Joseph,[3] about one per cent. of all so-called skin diseases, attacks both sexes equally and mainly between the ages of fifteen and thirty years. Both sides of the body are affected with equal frequency, but bilateral herpes zoster is extremely rare. There is an old superstition that should shingles entirely girdle the body it would be fatal. As a matter of fact, bilateral herpes zoster is usually due to serious disease of the vertebral column such as cancer or destructive Pott's disease. Epidemics of zoster occur in the spring and autumn, but sporadic cases appear with regularity throughout the year. There is a prodromal period of a few days with malaise and slight temperature. Pains, sometimes intensely neuralgic, burning, discomfort, uneasiness in varying degree, are felt in the part and suddenly the eruption appears. Commonly, pain and temperature then subside and the eruption runs its indicated course. Not infrequently, from infection of the eruption by various applications or the fingers, sloughs and even gangrene may result, but ordinarily under any cleanly management the blebs dry up and no serious scarring results.

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[1] Winfield. "N. Y. Med. Jour.," Aug. 2, 1902.

[2] "Brain," Dec., 1900.

[3] "Phila. Med. Jour.," Oct. 25, 1902.

The distribution of zoster is peculiar and significant. On the head and face it follows the distribution of the three main branches of the fifth nerve very closely in some instances and rarely invades the fields of two of them in one patient. More commonly it is confined to a patch within the territory of a single branch and located at the so-called maximal point of the segmental area outlined by Head (see page 59). On the trunk the eruption is either massed about the maximal points of pain in the segmental areas related to the spinal cord, or, as in the case illustrated, follows the limits of such area precisely. When occurring on the extremities, the eruption follows the longitudinal lines of the cord segments. The eruption does not follow peripheral nerves and their cutaneous distribution unless arising from injuries or inflammation of such nerves.

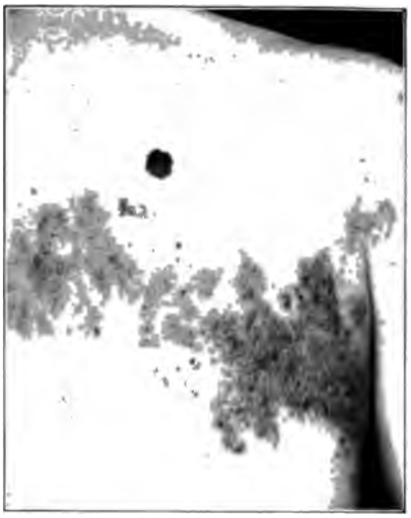

Fig. 119.—Herpes zoster confined to the fifth and sixth dorsal cord segments.

Diagnosis.—The diagnosis is very readily made, only vesicular eczema and simple herpes are likely to be confounded with it. The course of the malady will clear the problem as well as the anatomical relations of zoster which are not presented by eczema. Herpes occurring after arsenic and carbonic oxid gas poisoning and herpes associated with pneumonia, meningitis, malaria, and typhoid present identical changes in the ganglia, nerves, and skin.[1] In these forms the girdle distribution is not pronounced, and the skin eruption tends to be symmetrical, though typical zoster may be encountered in these associations.

Herpes simplex, affecting the lips and nose in coryza and gastro-intestinal intoxications, and herpes genitalis are still unclassified as to the participation of the root ganglia.

Treatment.—The treatment seems comparatively unimportant. An antiseptic local dressing, preferably a dry one, a free action of the bowels, and the elimination of any discoverable toxic or infectious element are the indications. If the pain or burning sensation is severe a one per cent. cocain ointment will give relief.

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[1] Howard, "Amer. Jour. Med. Sciences," Feb., 1903.

PART V.

DISEASES OF THE CORD PROPER.

CHAPTER I.

LOCALIZATION.

Anatomical Considerations.—The spinal cord reaches from the foramen magnum to the lower border of the body of the first lumbar vertebra. From its lower portion the lumbar and sacral nerve-roots extend to their several intervertebral foramina and make up the cauda equina, which occupies the dural sheath from the lumbar enlargement of the cord to the bottom of the sacral canal. The relations of the cord to the surface of the body and to the vertebral bodies and the spinous processes are shown in figures 120 and 121.

The cord is to be considered as made up of a number of similar and partly independent segments corresponding to the vertebral bodies, and each bearing a pair of spinal nerves. In addition it furnishes a longitudinal pathway for afferent and efferent nervous impulses. In early fetal life these cord-segments are in apposition with their corresponding vertebræ, but become gradually displaced upward as the spinal column outgrows the cord. At birth the lower end of the cord is opposite the third lumbar body. The nerve-roots, as a result of the unequal vertical growth of the cord and spine, become progressively longer from top to bottom, and in the same way the spinal-cord segment occupies a position relatively higher than its vertebral centrum. In the cervical region this amounts to the height of about one vertebral body, and in the dorsal region to about one and a half vertebral bodies. The five lumbar, five sacral, and one or two coccygeal segments are crowded into the lumbar enlargement below the upper level of the eleventh dorsal and above the second lumbar vertebral body.

In estimating the position of the various vertebral bodies we are guided by the subcutaneous tips of the spinous processes. It will be recalled that these have a downward direction in the cervical and dorsal region and overlap the body of the vertebra below. Thus the tip of the eighth dorsal spine is on a level with the ninth dorsal body. Reference to figure 121 will make this clear. It follows that the exit of the spinal root at the intervertebral foramen has only a relative relation to the cord-segment from which it originates. Cord-lesions, are, therefore, always above the level of their spinal-nerve symptoms.

Every spinal-cord segment possesses motor, sensory, and reflex functions besides vasomotor, visceral, and trophic activities. These are related to corresponding body-segments, which are shown in their cutaneous extent in figures 17 and 20. Their relation to the viscera has already been indicated (see p. 57).

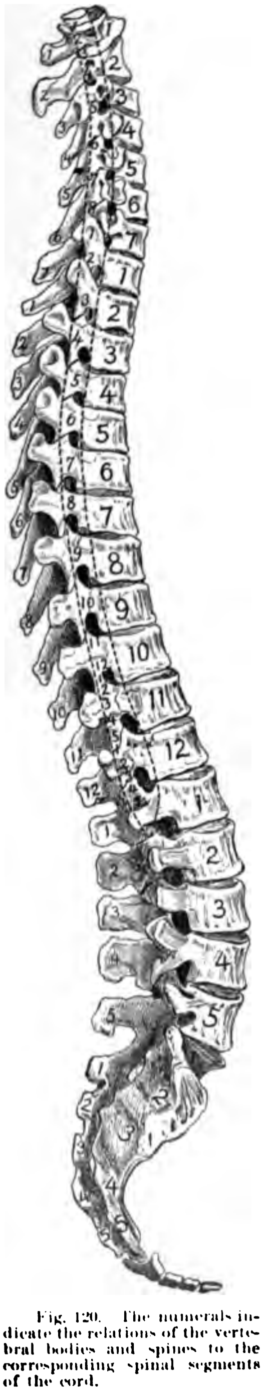

Fig. 120. The numerals indicate the relations of the vertebral bodies and spines to the corresponding spinal segments of the cord.

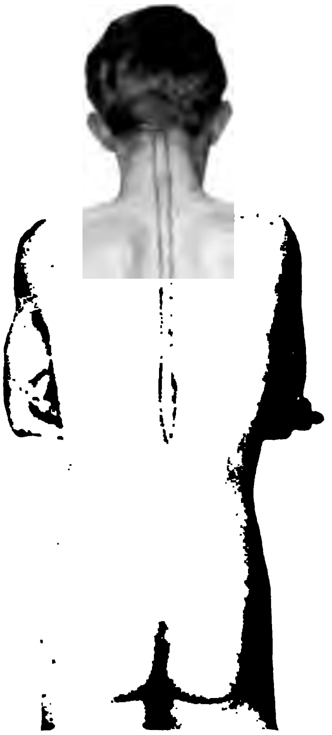

Fig. 121.—Showing the relation of the spinal cord to the body surface

Cross-sections of the cord show that the gray and white matter vary in proportionate area and outline at different levels (Fig. 123). The white matter is divided into numerous zones and tracts. One division is based on the embryological development of the various tracts (Fig. 124). From dissections, physiological experiments, and especially from the results of morbid processes, we have still further division of the tracts. These are shown in figure 125 for the ascending degenerations which follow complete division of the cord, and for the descending degenerations after similar lesions.

The H-shaped cross-section of the gray matter shows numerous cell-bodies. Of these the anterior cornual groups are the best understood and contain the cell-bodies of the lower motor neuron through which motor, trophic, and vasomotor influences are exercised over the muscles, bones, and skin. The central canal is situated in the gray commissure. The essential elements of the cord are supported by fibrous tissue which is continuous with the penetrating septa of the pia mater.

The circulation in the cord is one of its most important anatomical features. Many of the cord diseases are due to vascular lesions or infections, and both their vertical distribution and lateral outlines in transverse sections may be limited to the corresponding arterial fields.

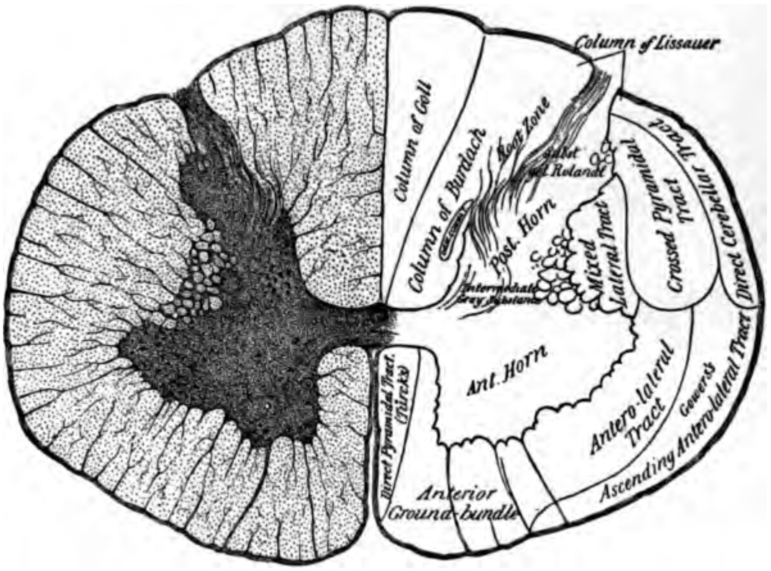

Fig. 122.—Cross-section of cervical spinal cord, showing its anatomical subdivisions (Schaefer).

The arterial supply of the cord consists of two systems, anterior and posterior. The *anterior spinal artery,* arising within the skull by two roots from the vertebrals, extends the entire length of the cord at the anterior fissure (Fig. 127). It is reinforced by branches from the intercostal, lumbar, and sacral arteries, which follow the corresponding nerves into the spinal canal and accompany the nerve-roots to the cord. The anterior spinal artery gives off about three hundred branches, called *anterior median arteries,* which penetrate the anterior fissure at a right angle to the parent stem. At the commissure they enter the cord and without dividing turn toward the right or left anterior horn. At the neck of the horn the artery divides into an anterior branch to the anterior horn and a posterior branch which is distributed to the neck and to a portion of the posterior horn. A branch is also given off in a

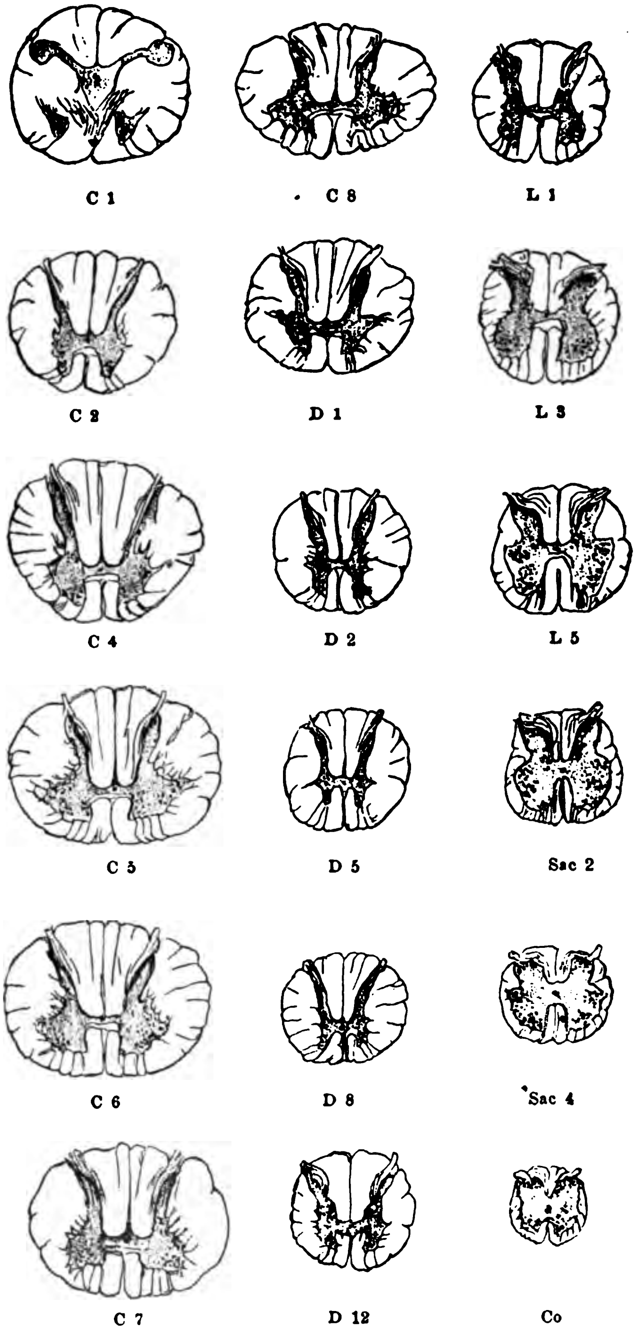

Fig. 123.—Transverse sections of the cord at various levels, to show the relative variations in gray and white matter.

figure 125 for the ascending degenerations which follow complete division of the cord, and for the descending degenerations after similar lesions.

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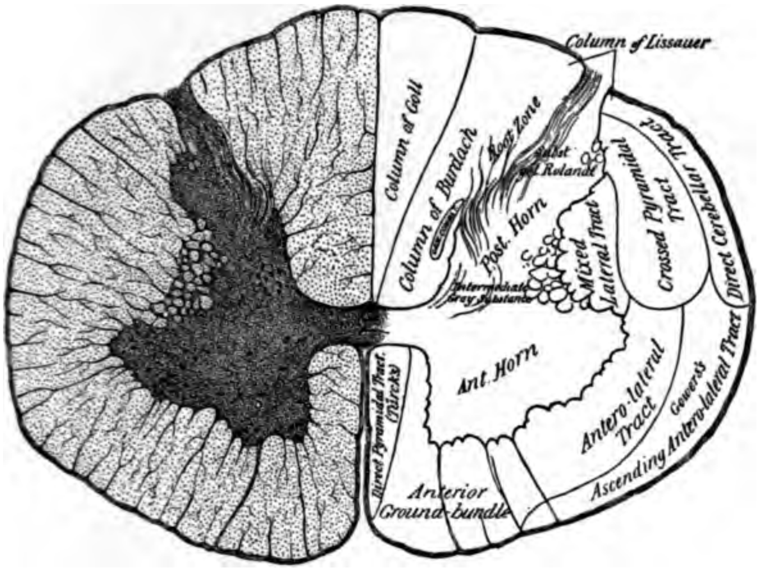

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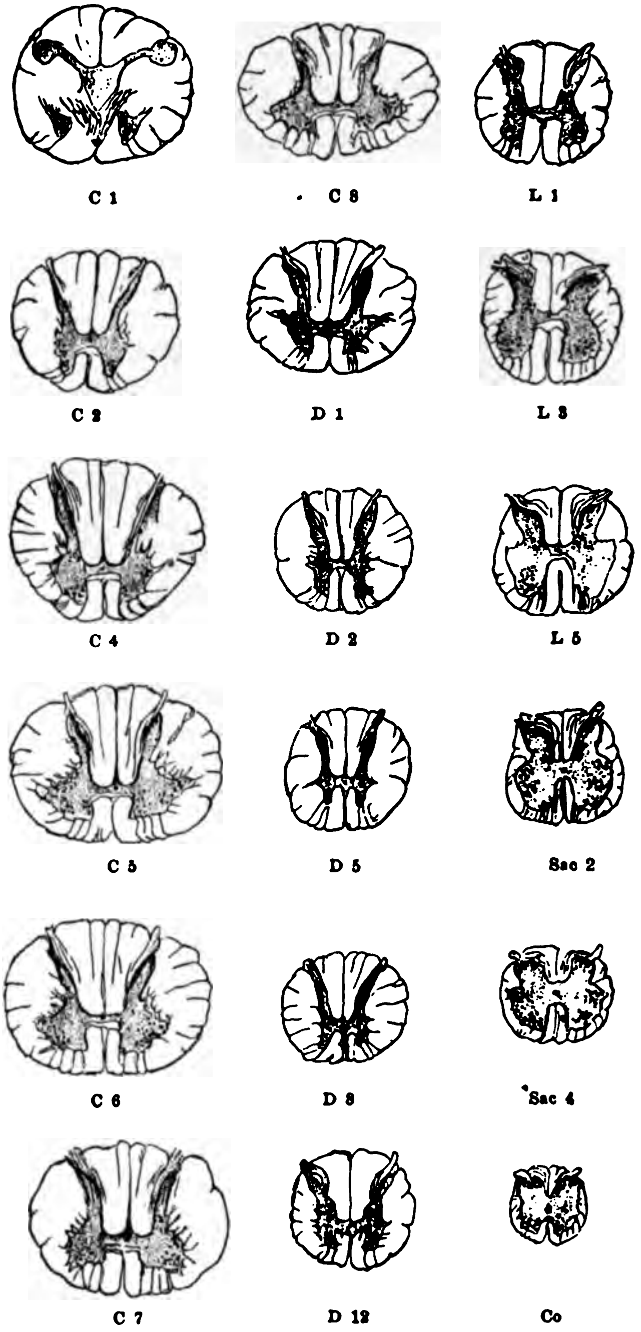

Fig. 123.—Transverse sections of the cord at various levels, to show the relative variations in gray and white matter.

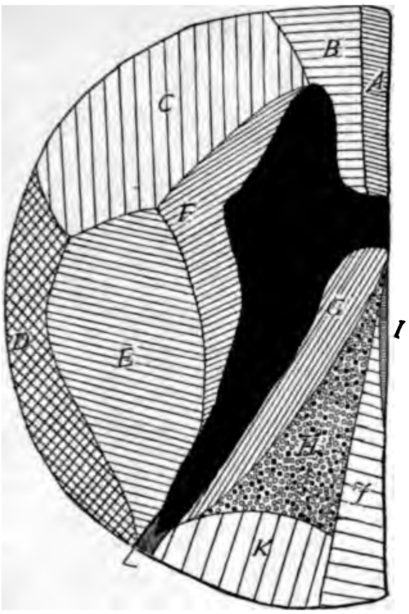

Fig. 124.—**Divisions of the cross-section of the cord based on Flechsig's study of their development.** *A*, **Direct pyramidal tract**; *B*, **fundamental fasciculus of the anterior column**; *C*, **rest of lateral column.** *D*, **direct cerebellar tract**; *E*, **crossed pyramidal tract**; *F*, **lateral limiting zone**; *G*, **anterior root zone**; *H*, **middle root zone**; *I*, **median zone**; *J*, **column of Goll**; *K*, **postero-internal root zone**; *L*, **postero-external root zone or zone of Lissauer (Marie).**

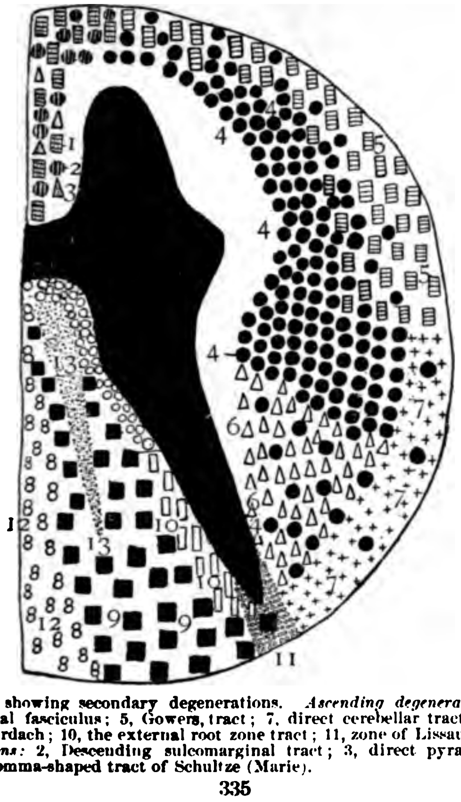

Fig. 125.—**Scheme showing secondary degenerations.** *Ascending degenerations:* 1, **Fibers of the ascending sulcomarginal fasciculus**; 5, **Gowers, tract**; 7, direct cerebellar tract; 8, cornucommissural **tract**; 9, **column of Burdach**; 10, **the external root zone tract**; 11, zone of Lissauer; 12, column of Goll. *Descending degenerations:* 2, Descending sulcomarginal tract; 3, direct pyramidal tract; 6, **crossed pyramidal tract**; 13, **comma-shaped tract of Schultze** (Marie).

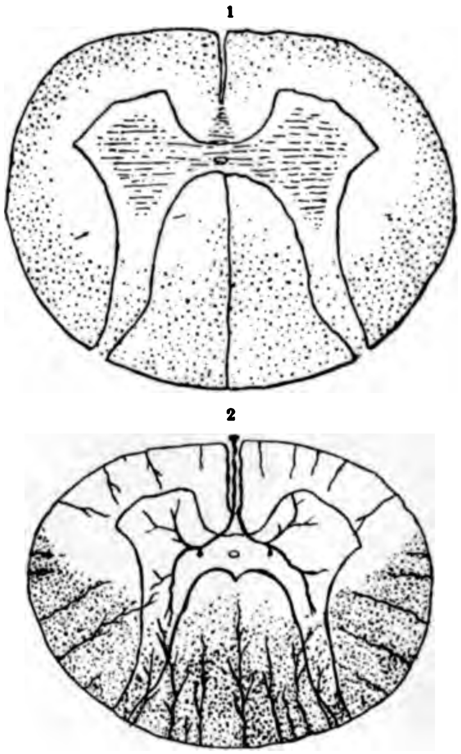

Fig. 126.—1, Section of the cord showing division into three arterial districts (diagrammatic). Part supplied only by the anterior median and its branches is shaded with parallel lines. Part supplied only by the peripheral arteries is shaded with dots. Part supplied by both systems of arteries is unshaded. 2, Transverse section of cord showing distribution of anterior and posterior arteries (modified after Marie). Part supplied by posterior arterial system shaded with dots. Part supplied by anterior system unshaded (Williamson).

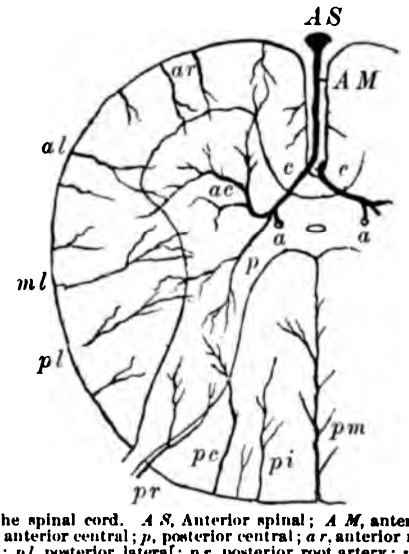

Fig. 127.—Arteries of the spinal cord. *A S*, Anterior spinal; *A M*, anterior median; *c c*, commissural; *a a*, anastomotic; *a c*, anterior central; *p*, posterior central; *a r*, anterior root arteries; *a l*, anterior lateral; *m l*, median lateral; *p l*, posterior lateral; *p r*, posterior root artery; *p c*, posterior cornual; *p i*, intermediate septal; *p m*, posterior median (modified after Obersteiner).

vertical direction communicating with the next similar artery, and forming a longitudinal chain in the gray matter (Fig. 128). The anterior spinal artery, from its lateral ramifications on the surface of the cord, also supplies the white matter in the periphery of the ventral half of the cord.

The posterior arterial system of the cord supplies its dorsal half. The two *posterior spinal arteries* arise separately from the vertebrals and, coursing around the medulla, extend the entire length of the cord just outside the posterior nerve-roots. Like the anterior spinal artery, they receive numerous supply branches from the intercostal and lumbar arteries. They give off *anastomotic branches* which unite with those of the anterior system on the surface of the cord, and also present a chain of anastomosing branches on the posterior median line of the cord. From these branches small twigs penetrate the cord supplying the gray matter of a portion of the posterior horn and the posterior half of the white matter of the cord (Figs. 127 and 128). The arterial twigs entering the cord are of the terminal variety, and therefore do not anastomose. The territories of the two systems are not entirely independent,, as the borders of their irrigation fields overlap and the adjacent white and gray matter receive arteries from both sources. Three arterial districts are thus constituted: (1) That supplied only by the anterior system; (2) that supplied only by the posterior system, and (3) that irrigated by both systems (Fig. 126).

It will be apparent from these facts that arterial disease may induce lesions in the posterior half of the cord, or in the anterior half. Further, the infection or obliteration of a single anterior median artery will practically destroy the corresponding anterior horn.

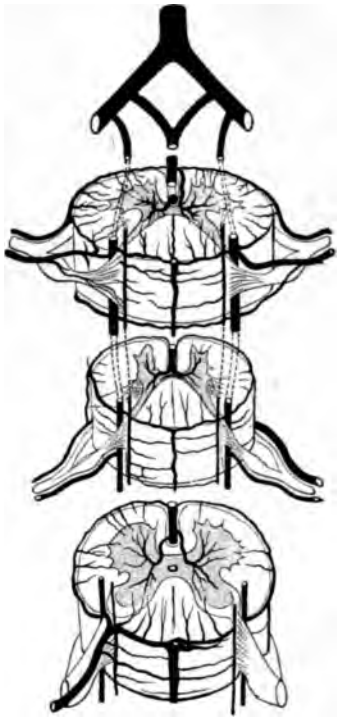

Fig. 128.—Scheme to show circulation in cord segments and in vertical directions (Brissaud).

The circulation in the cord, as a whole, presents some mechanical conditions that are significant. The spinal arteries are the longest of their size in the body, and, owing to their course, are not subject to the direct impact of cardiac impulses. In this respect they are very different from those of the brain. Arterial pressure is also slight, and the venous outlet into the plexuses about the dura is not an advantageous one in the erect position. Gravity impedes the return circulation as well as the supply. This is most marked at the lower end of the cord. The long course the lumbar and sacral arteries have to pursue in an upward direction along the corresponding nerve-roots in the cauda still further reduces the circulatory qualifications of this end of the cord.

The arteries in the cord are provided with perivascular lymph-sheaths, which are continuous with the arachnoid meningeal spaces. Venæ comitantes accompany the arterial branches and empty into posterior and anterior plexiform venous chains on the surface of the cord, discharging in turn into the extradural plexuses. Regurgitation from the extradural veins is probably impossible.

The *posterior roots* bear a ganglionic enlargement situated just outside the dura, except on the lowest pairs, where it is within the sheath. Its function is trophic for the afferent tracts in the cord.

Vertical Localization of a Cord-lesion.—Cord-lesions are irritative or destructive and are manifested by corresponding symptoms in the periphery to which the injured cord-segments are anatomically related and to which their peripheral nerves are distributed. Irritative lesions are marked by sensory exaggeration, such as hyperesthesia and pain, by cramps and increased reflexes. Anesthesia, paralysis, and abolished reflexes result from destruction of a cord-segment. Trophic loss and vasomotor paralysis may also ensue, but become apparent at a later date. The symptoms of a cord-lesion embrace: (1) Those due to the derangement of the injured segment; (2) those resulting from disturbances in the conduction attributes of the cord, and (3) those arising from the secondary degenerations in the cord. These are variously combined, depending upon the extent of the lesion in the cross-section of the cord and its age.

Motor Symptoms.—A completely disabling injury falling upon any cord-segment produces *paralysis* in the muscles supplied by that segment. It will be noticed, in the following table, that muscular actions are functionally grouped in the cord and extend in a vertical direction through several segments. Single muscles are, therefore, practically never alone paralyzed by spinal lesions. Such a circumscribed palsy, or one confined to a group of muscles supplied by a single nerve-trunk, at once declares the peripheral character of the lesion. Not only are the muscles supplied by the injured segment paralyzed, but all muscles represented in the cord below the lesion are withdrawn from voluntary control through the division of their motor paths in the upper neurons, which traverse the cross-lesion by way of the pyramidal tracts. A paraplegia is thus induced.

If the lesion is not a destructive one, but irritative in its effects, *spasms* and *rigidity* may be present. These are usually attended by clonus in the large muscles whose tendons pass over joints; they are present in all the levels below the upper limit of the lesion, but more especially in the lower limbs.

In late cases of partial cross-lesions the rigidity and *spastic features* are well developed. The lower limbs are held rigidly extended or, less frequently, sharply flexed. Any attempt to move them causes clonic trembling, which may finally end in a sharp pulling up of the leg with a "jack-knife"-like jerk, or the thighs may be held so rigidly by the muscles of the pelvic girdle that if one limb is raised from the bed by the foot the other follows with it. *Contractures* finally develop: (1) By the paralysis of one group of muscles and the unopposed overaction of antagonists; or (2) by the overirritation of the motor mechanism of certain muscles or muscle-groups; or (3) by structural changes in the muscles, causing a tendinofibrosis, with its characteristic retraction. Only when dealing with the third form is tenotomy permanently useful, as in the other varieties the activity of the muscular masses promptly reproduces the conditions sought to be relieved. In the structural variety the distorted joint usually has a certain limited range of free motion, but is then abruptly stopped by the retracted, unyielding tendons, which stand out prominently. On the other hand, the muscular contractures, due to overstimulation in which fibroid changes have not taken place, usually yield slowly or, perhaps, jerkily to efforts tending to extend them.

MOTOR AND REFLEX FUNCTIONS OF THE SPINAL-CORD SEGMENTS (AFTER STARR AND EDINGER).

SEGMENT.	MUSCLES.	REFLEXES.
Cervical	Sternomastoid.	
	Trapezius.	
2-3	Scaleni.	
	Small rotators of head.	
	Diaphragm.	
	Lev. ang. scap.	Dilatation of pupil by irritating side of neck, 4-7 cervical.
	Rhomboids.	
4	Spinati.	
	Deltoid.	
	Supinat. long.	Scapular reflexes, 5 C.-1 D.
	Biceps.	Supinat. long., 5 C.
5	Supinat. brev.	
	Serrat. mag.	
	Pectoralis (clav.)	Biceps, 5-6 C.
	Teres minor.	
6	Pronators.	Posterior wrist, 6-8 C.
	Brachialis ant.	
	Triceps.	
7	Long extensors of wrist and fingers.	Anterior wrist, 7-8 C.
	Pectoralis (costal).	
	Latiss. dorsi.	Palmar, 7 C-1 D.
	Teres maj.	
	Long flexors, wrist and fingers.	Epigastric, 4-7 D.
8	Extensors of thumb.	
Dorsal 1	Intrinsic hand-muscles.	Abdominal, 7-11 D.
2-12	Dorsal and abdominal muscles.	
Lumbar	Abdominal muscles.	Cremaster, 1-3 L.
1	Iliacus.	
	Psoas.	Patellar, 2-4 L.
2	Sartorius.	Bladder, 2-4 L.
	Flexors of knee.	
	Quad. femoris.	
3	Int. rotators of thigh.	
	Adductors of thigh.	Rectal, 4 L.-2 S.
	Abductors of thigh.	
4	Tibialis ant.	Gluteal, 4-.5 L.
	Calf-muscles.	
5	Ex. rotators of thigh.	
	Extensors of toes.	Achilles, .5 L.
	Peronei.	
Sacral 1-2	Long flex. of toes.	Plantar, 1-2 S.
	Intrinsic foot-muscles.	Anal, Virile, 3-5 S.
3-5	Perineal muscles.	

Sensory Symptoms.—A cross-lesion destroys cutaneous sensation in the segmental area corresponding to the lesion and in all parts below, the first directly, the second by interruption of conduction. The distribution of *anesthesia* is practically the best guide to the upper level of the lesion. In the diagrams shown on pages 55 and 58 it will be seen that the cutaneous areas of the skin do not correspond exactly to the cutaneous distribution of the spinal nerves. This variation will often serve to differentiate between a cord-lesion and one of the nerve-roots. Root-lesions cause areas of sensory disturbances that coincide with the cutaneous distribution of the nerves arising from them. In the trunk, for instance, division of the cord produces an anesthesia whose upper level is practically in a horizontal plane, while root-lesion anesthesia or hyperalgesia follows the intercostal nerves and spaces. The territories supplied by the intercostal nerves overlap decidedly, so that destruction of a single nerve may manifest no sensory loss. It requires that at least two should be divided to produce an anesthetic patch or belt.

The upper border of a spinal lesion is usually further indicated by a zone of *hyperesthesia* due to the irritant action of the lesion on sensory paths within the cord or upon the posterior nerve-roots. This is described by the patient as a band or girdle-like sensation about the trunk and as tightness in the limbs. Its vertical extent corresponds to the irritating influence, but rarely does it exceed two segmental areas. A light touch in this hypersensitive zone causes a feeling as of pins and needles, of pricking, of burning, of tingling, or of a thrill, and is not a pure exaggeration of sensation, but a perversion of it. Very severe pain is usually absent in pure cord lesions, but the girdling sensation is often described as painful; and if the posterior roots are injured, as in Pott's disease or through meningeal inflammation, the pains are intense, and often darting in character. *Spontaneous sensations* are frequent, and patients often attempt to describe peculiar feelings of an unnatural sort which they may locate in areas that are absolutely insensitive to external stimulation. They may be caused by the irritation of the conduction tracts at the upper level of the lesion, and are then referred to the peripheral sites from which they would naturally arise.

In lesions of lesser degree sensation may not be much affected, even when motion is abolished, or the general sense of touch may be broken up so that temperature and painful impressions are not recognized. Tactile impressions, in the same way, may fail to arouse the sensorium when more energetic and painful impressions still traverse the injured pathways.

The reflexes furnish very valuable evidence not only as to the upper level of the cord lesion, but sometimes as to its vertical extent. The cord lesion that destroys the portion of a reflex are within the cord obliterates that reflex but does not abolish the reflexes below its own level unless the cord is entirely and completely divided. It is now fairly well established that complete division of the cord in man extinguishes all cord reflexes below that level.[1] On the contrary, if the division is incomplete, the lower reflexes may at first be enfeebled, but within a few weeks show much exaggeration. In a case showing such increased reflexes the absence of one or more at a given level points to the diseased portion of the cord. Again, the upper level of abolished reflexes usually coincides with that of anesthesia, and both focalize the lesion. In other cases the extension and invasion of cord disease may be traced by the successive disappearance of spinal reflexes; its recession, by their reappearance.

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[1] Collier, "Brain," Spring, 1904.

Trophic Conditions.—As the trophic centers for muscles correspond with their motor spinal centers in the anterior horns, a lesion which destroys this portion of the gray matter of a cord's segment induces atrophy in the related muscles. This *atrophy,* like the palsy arising from a cord lesion, has a functional distribution that depends upon the association of muscular representation in the cord. By reference to the table, page 339, it is evident that all of a large muscle need not be involved, and that groups of muscles innervated by different nerve trunks, but centrally associated, may be thus selected. The extent of the wasting is limited by the vertical dimensions of the lesion. The muscles innervated from the cord above and below the destructive process are spared, and retain their nutritional supply and their electrical responses. The wasted muscles lose tone early, and shortly thereafter present the electrical reaction of degeneration. Atrophic loss is best seen in the muscles of the extremities; next in those of the shoulder and pelvic girdles. In the thoracic and abdominal regions the wasting is only perceptible when several adjacent cord segments are diseased.

Slight *atrophic disturbances* appear in the skin, analogous to those in neuritis and confined to the area related to the injured cord segments. In the case of irritative lesions, such as acute myelitis and cord hemorrhage, the dystrophic condition may be acute and intense. Under the incitement of comparatively slight pressure or other superficial irritation, herpetoid eruptions and acute bedsores form in a few hours. These occur over the sacrum, heels, malleoli, and trochanters by preference, but may occur wherever the bones are subcutaneous and the trophic control disturbed. The use of counterirritation in the mildest form, and even of frictions with the hand, may provoke them. Hot bottles that would otherwise cause no injury may, under these circumstances, induce most serious local effects and deep destruction of tissue.

Vasomotor disturbance in some degree is usually present, and consists, ordinarily, at first of a tendency to vascular dilatation and increased warmth. The so-called *tache cérébrale* is easily provoked below the lesion. In cases of long standing the skin is livid and cold, frequently with increased perspiration. Lesions in the cervical region often cause flushing and perspiration on the side of the neck and face, and may reduce the heart-beats to forty or even to twenty a minute. Dorsal lesions, on the other hand, are sometimes attended by a persistently rapid pulse. These vasomotor disturbances are frequently attended by an increase of body temperature in lesions in the upper dorsal and lower cervical regions, but it is often difficult to exclude pyrexia, due to the infectious nature of the disease or to intestinal or vesical disturbance resulting from it.

Visceral symptoms are usually not pronounced, but the secretions in the *alimentary canal* and its muscular activity are frequently disturbed. Constipation is the rule, and fermentation of the stomach and intestinal contents, with gaseous disturbance and great abdominal distention, is very common. Vomiting and difficulty in swallowing occur in lesions of the cervical cord. The anal and vesical sphincters are usually disturbed. When the lesion involves their reflex centers in the lumbar cord, complete relaxation and incontinence ensue; but if the lesion is above their spinal centers, voluntary control alone is lost. They then act automatically, and the corresponding viscera are unconsciously evacuated at intervals. The examining finger readily provokes the anal sphincteric contraction in this condition, which is not the case if its center is destroyed. There is a tendency to fecal and *urinary retention*, dependent, in part, upon the lack of power in the abdominal muscles. In the case of the bladder this leads to distention by increased residuum and weakening of the detrusor, and the dilatation of the viscus may become extreme. The result is usually a cystitis, which is often precipitated by the use of a septic catheter. Damage to the *kidneys* arises both from the back-pressure of urine and the propagation of inflammation up the ureters. The table of symptoms in disabling but not absolutely destructive transverse cord lesions (see pages 343–349) is based upon the exhaustive work of Wichman.[1] If the entire cross-section is absolutely destroyed, the symptomatology is the same excepting that there is complete absence of muscle reflexes below the lesion.

Horizontal Localization of a Cord Lesion.—Many spinal cord lesions are more or less circumscribed in transverse area, and give rise to widely different symptoms as they affect the various physiological divisions of the cord. Some cord diseases are symmetrical, and both sides of the transverse section show identical conditions. Others are unilateral, and the two sides show different states, not only at the level of the lesion, but also in the levels affected by the secondary ascending and descending degenerations.

Lesions of the *pyramidal tracts* produce motor paralysis below the level affected and induce a spastic condition in the paralyzed area, marked by increased muscle tonus, exaggerated reflexes, rigidity, and contractures. These tracts degenerate downward.

Disease of the *posterior columns* is marked by sensory disturbance, especially of those elements of touch related to pressure and to the muscle and joint sensations. Ataxia results. The temperature and pain sensations are also usually diminished, and all forms of cutaneous impressions are delayed in reaching the brain. The muscle reflexes, especially the deep tendon reflexes, are abolished or greatly diminished. An upward degeneration in the postero-internal column ensues.

When the *anterior horn* is affected motor paralysis occurs, but only in the muscles which are supplied by the large cells actually involved in the morbid focus. The mucles also atrophy. If the process is acute, paralysis takes place at once and atrophy gradually develops. In very insidious lesions paresis and atrophy develop at an equal pace, and fibrillary twitchings are usually present. Reflexes are abolished by lesions of the anterior horns and vasomotor paralysis is induced in the field related to the cornual disease. Degeneration descends the lower neurons arising from the diseased portion of the cord, and the reaction of degeneration is presented in nerve and muscle.

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[1] "Die Rückenmarksnerven und ihre Segmentbezüge," Berlin, 1900.

TABLE OF SYMPTOMS IN CROSS-LESIONS OF THE CORD.

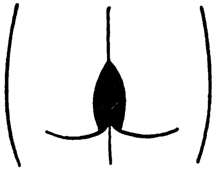
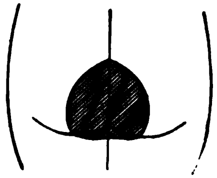
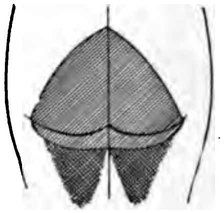
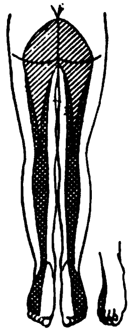
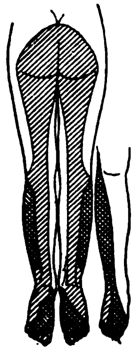
Lesion. Segment.	Motor Conditions.			Reflex Conditions.		Sensory Conditions.	
	Paralysis.	Paresis.	Actions Lost or Impaired.	Absent.	Increased in Partial Lesions.	Anesthesia Marked by Diagonal Lines.	Hypesthesia Marked by Crossed Lines.
V. Sacral.	None.	Coccygeus.	Elevation of coccyx.	None.	None.		
IV. Sacral.	Coccygeus.	Levator ani. Sphincter ani. Detrusor urinæ. Transversus perinei. Erector penis. Compressor urethræ.	Elevation of coccyx. Elevation of anus. Sphincter ani. Ejection of urine. Vaginal constriction.	Erection of penis diminished.	None.		
III. Sacral.	Sphincter ani. Levator ani. Detrusor urinæ. Transversus perinei. Erector penis. Compressor urethræ.	Rectum.	Defecation disturbed. Retention of urine, later followed by dribbling. Ejaculation lost. Erection possible, but paretic. [Testicle sensitive to pressure.]	Ejaculation lost. Erection diminished. Tendo Achillis.	None.	Rider's breeches form.  Perineum. Anus. Scrotum (posterior inferior part or labia). Penis. Thigh, uppermost posterior part hypesthetic.	

TABLE OF SYMPTOMS IN CROSS-LESIONS OF THE CORD.—(*Continued.*)

LESION. SEGMENT.	MOTOR CONDITIONS.			REFLEX CONDITIONS.		SENSORY CONDITIONS.	
	Paralysis.	Paresis.	Actions Lost or Impaired.	Absent.	Increased in Partial Lesions.	Anesthesia Marked by Diagonal Lines.	Hypesthesia Marked by Crossed Lines.
II. Sacral.	Sphincter ani. Levator ani. Detrusor urinæ and other muscles as in III. Sacral.	Pyriformis. Obturator internus. Gemellus superior. Gluteus maximus. Biceps femoris. Gastrocnemius. Soleus. Tibialis posticus. All the small muscles of foot.	Outward rotation thigh. Retraction thigh. Flexion of knee. Plantar flexion of foot. Standing on the toes. Raising inner margin of foot. Defecation } as in III. Sacral. Retention }	Ejaculation. Erection. Plantar weakened.	None.	Anesthesia of genitalia, except base of penis and scrotum; hypesthetic	
I. Sacral.	Muscles of anus. Muscles of bladder. Muscles of genitals. Pyriformis. Adductor hallucis. Flexor hallucis brevis. I-IV, Dorsal interossei. I-III, Plantar interossei. III-IV, Lumbricales. Abductor minimi digiti. Opponens minimi digiti.	Gluteus maximus. Obturator internus. Gemellus superior. Gluteus medius. Gluteus minimus. Biceps. Semimembranosus. Semitendinosus. Popliteus. Gastrocnemius. Soleus. Tibialis posticus. Peroneus longus. Peroneus brevis. Flexors of toes. Extensors of toes.	Retention of feces. Retention of urine, or dribbling. Erection and ejaculation impossible. Outward rotation of thigh impaired. Internal rotation impaired. Flexion of knee difficult. Plantar flexion of foot. Raising inner margin of foot. Raising outer margin, and dorsal flexion of foot. Flexion and extension of toes, adduction of great toe, abduction of little toe, etc.	Plantar weakened. Achilles-tendon reflex. Ejaculation. Erection. Micturition. Defecation. Gluteal.	None.		

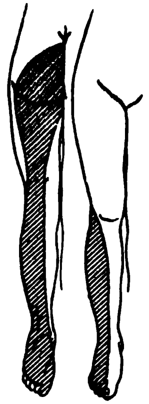
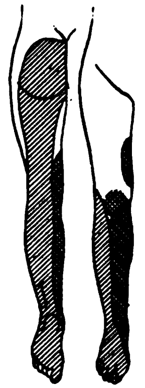
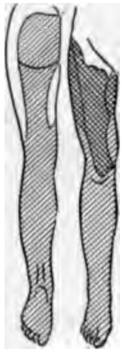
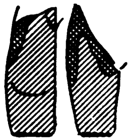
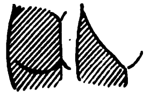
V. Lumbar.	Muscles of anus and rectum. Muscles of bladder. Muscles of genitals. Pyriformis. Obturator internus.	Gemellus superior. Gemellus inferior.	Defecation. Micturition delayed, dribbling. Erection, ejaculation impossible. Outward rotation of thigh very difficult.	Ejaculation. Erection. Micturition. Defecation. Gluteal.	Plantar. Tendo Achillis.	
		Gluteus medius. Gluteus minimus.	Inward rotation impaired.			
	Biceps femoris.	Semimembranosus. Semitendinosus.	Flexion of knee difficult.			
		Gluteus maximus. Tensor fasciæ femoris.	Retraction of thigh very difficult.			
		Gastrocnemius. Soleus.	Flexion of foot barely possible.			
	Flexors of toes.		Flexion of toes impossible.			
		Extensors of toes.	Extension of toes weak, except great toe, which may be dorsally flexed.			
		Tibialis anticus.	Raising inner margin of foot difficult.			
	Peroneus longus. Peroneus brevis.		Raising outer margin of foot impossible.			
IV. Lumbar.	Muscles of rectum and anus. Muscles of bladder. Muscles of genitals.		Defecation. Micturition delayed, dribbling. Erection, ejaculation impossible.	Patellar may be wanting.	Plantar.	
	Obturator internus. Pyriformis. Gemelli.	Obturator externus.	Outward rotation of thigh weak.			
	Gluteus medius. Gluteus minimus.		Inward rotation impossible.			
	Gluteus maximus.		Retraction of thigh impossible.			
	Biceps femoris. Semimembranosus. Semitendinosus. Popliteus.		Flexion of knee lost.			
	Gastrocnemius. Soleus.		Plantar flexion of foot lost.			
	Flexors of toes. Extensors of toes.		Flexion and extension of toes lost.			
	Peroneus brevis. Peroneus longus.		Raising outer margin of foot.			
	Tibialis anticus.		Raising inner margin.			
		Rectus femoris. Vastus externus. Vastus internus.	Extension of thigh weak.			
		Adductor magnus. Adductor brevis. Adductor minimus. Gracilis.	Adduction difficult.			

TABLE OF SYMPTOMS IN CROSS-LESIONS OF THE CORD.—(*Continued.*)

LESION. SEGMENT.	MOTOR CONDITIONS.			REFLEX CONDITIONS.		SENSORY CONDITIONS.
	Paralysis.	Paresis.	Actions Lost or Impaired.	Absent.	Increased in Partial Lesions.	Anesthesia Marked by Diagonal Lines. Hypesthesia Marked by Crossed Lines.
III. Lumbar.	Muscles of anus, bladder, and genitals. Outward rotators thigh. Inward rotators thigh. Retractor (flexor) thigh. Flexors of knee. Plantar flexors of foot. Flexors of toes. Extensors of toes. Extensors of foot. Vastus externus.	Vastus internus. Rectus femoris. Crureus. Adductors of thigh. Flexors of thigh at the hips.	Extension of leg almost impossible. Psoas and iliacus can lift the leg somewhat, however.	Patellar. Cremasteric.	Ankle-clonus may exist.	
II. Lumbar.	Paralysis of all muscles of lower extremity except psoas.	Psoas.	All motions lost. [Testicle not sensitive to pressure.]	Patellar. Achilles. Cremasteric.	Achilles may be increased. Plantar.	 Total anesthesia of lower extremity except territory of the ilio-inguinal and external cutaneous, which is hypesthetic.
I. Lumbar.	Total paralysis of whole lower extremity, psoas included.			Cremasteric. Achilles.	Patellar none or increased. Plantar. Ankle clonus.	

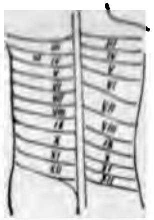
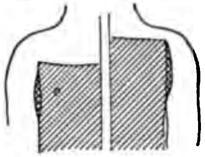
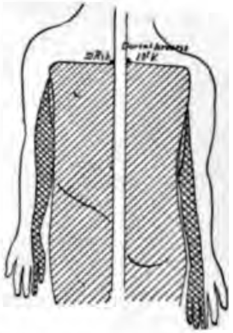
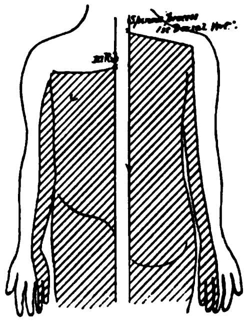
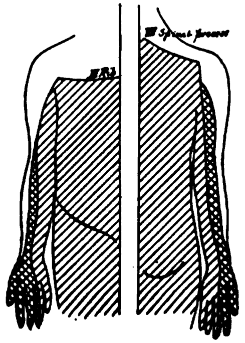
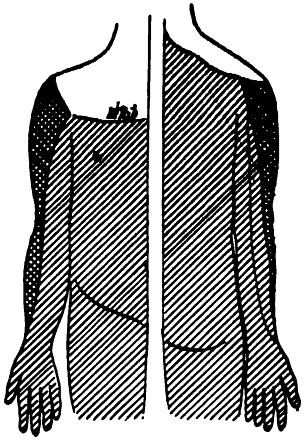
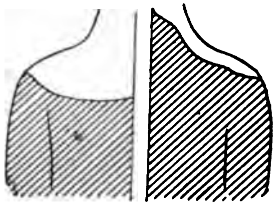
XII-III. Dorsal.	Paralysis of lower extremity, and gluteal region. Paralysis of abdominal and dorsal regions gradually added as the site of the lesion ascends.		Paralysis of muscles of respiration causes diaphragmatic breathing and dyspnea.	In complete lesions epigastric reflex and abdominal reflex may be abolished. All below lost in complete division of cord.	Increased in incomplete lesions. Patellar. Cremasteric. Achilles. Plantar.	 Anesthetic area begins, as a rule, two segments below site of lesion, these two intervening segments being hypesthetic.
II. Dorsal.	As in III. Dorsal.			All below lost in complete division of cord.	All subjacent reflexes.	
I. Dorsal.	All muscles of trunk and lower extremities.	Flexors of fingers. Muscles of the little finger. III and IV Interossei. Lumbricales. Pronator quadratus. Lower part pectoralis major. Lower part pectoralis minor.	Weakness in flexion of fingers. Pronation disturbed.	Oculopupillary symptoms. All below lost in complete division of cord.	All below increased.	

TABLE OF SYMPTOMS IN CROSS-LESIONS OF THE CORD.—(*Continued.*)

LESION. SEGMENT.	MOTOR CONDITIONS.			REFLEX CONDITIONS		SENSORY CONDITIONS.	
	Paralysis.	Paresis.	Actions Lost or Impaired.	Absent.	Increased in Partial Lesions.	Anesthesia Marked by Diagonal Lines.	Hypesthesia Marked by Crossed Lines.
VIII. Cervical.	Paralysis muscles of trunk and lower extremities. Abductor of little finger. Adductor of thumb. Flexors of the little finger. Opponens minimi digiti. III and IV Interossei. Lumbricales.	Flexors of the little finger. Opponens minimi digiti. Flexor sublimis digitorum. Flexor profundus digitorum. Flexor carpi ulnaris. Extensors of the thumb and fingers. Extensor carpi ulnaris. Triceps (slight). Latissimus dorsi (lowest part). Pectoralis major. Pectoralis minor. Scalenus medialis. Scalenus posterior.	Hand weak. Extension of arm. Int. rotation and retraction of arm. Adduction of arm.	Oculopupillary symptoms. All below lost in complete division of cord.	All below increased.		
VII. Cervical.	Lower extremities and trunk. Flexor profundus digitorum (ulnar side). Flexor carpi ulnaris. Small hand-muscles. Pronator quadratus.	Extensors } Flexors } of thumb. Abductors } Extensor indicis. Extensors of the fingers (movements barely possible). Supinator longus. Biceps (very slightly paretic). Triceps. Pectoralis major Serratu magnus (slight). Latissimus dorsi. } Teres major. }	Hand very weak. (“Winged” scapulæ.) Retraction and inward rotation of the arm.	Arm reflexes. Forearm reflexes. Palmar reflex. All below lost in complete cord division.	All below increased.		

VI. Cervical.	Muscles of lower extremity and trunk.			Arm-reflexes. Extensor forearm reflexes. All below lost in complete cord division.	All below increased.	
	Muscles of fingers (including thumb) and hand.		Movements of fingers and thumb impossible.			
	Triceps.	Coracobrachialis. Biceps. Brachialis anticus. Supinator brevis.	Extension of forearm. Flexion of forearm weak. Supination very weak.			
	Pectoralis major. Latissimus dorsi. Teres major.		Adduction of arm and inward rotation.			
	Infraspinatus.		Adduction, retraction, and external rotation.			
	Serratus magnus.		"Winged" scapula.			
		Deltoid.	Raising of arm.			
		Scaleni. Splenii. Deep head and neck muscles.	Rotation of head.			
			Fatal in a few days or weeks.			
V. Cervical.	Muscles of lower extremity and trunk. All the muscles of the arm, forearm, hand, and fingers; even the deltoid, coracobrachialis, and brachialis anticus.			Scapular. Tendon reflexes of paralyzed muscles. All below lost in complete cord division.	All below increased.	
		Levator anguli scapulæ. Scaleni.	Shoulders raised with difficulty. Rotation and flexion of head.			
	Deep cervical muscles. Intercostals.	Diaphragm (because of filaments from V. cervical segment to the phrenic nerve), or spread of injury from 5th to 4th cervical segment.	Dyspnea.			
		Trapezius and sternocleidomastoid are intact.	Fatal in a few hours or days.			
IV-I. Cervical.	Total cross-lesions from the fourth cervical segment upward are rapidly fatal, because of complete paralysis of the diaphragm and intercostals.					

Disease of the *posterior horn* is marked by sensory disturbance or anesthesia in a given area, such as follows a lesion of the posterior column.

Lesions of the *posterior roots* cause anesthesia if complete; hyperalgesia and radiating pain if irritative.

Lesions of the *anterior roots* produce the same results as lesions of the anterior horns.

Lesions arising within or immediately about the *central canal*, as in syringomyelia, produce a peculiar dissociation of touch sensations, so that painful and thermal impressions are not properly recognized while tactile or contact impressions remain practically unaffected. Joint dystrophies are often added.

A lesion that divides *one lateral half* of the cord gives rise to the Brown-Séquard syndrome (see p. 55).

In many cord diseases two or more physiological divisions are symmetrically affected. In amyotrophic lateral sclerosis we have, added to the rigidity, myotatic irritability, and contractures that mark disease of the lateral tracts, a progressive muscular atrophy that is due to the lesion of the anterior horns. Ataxic paraplegia is marked by symptoms in both the lateral tracts and the posterior columns, and we find ataxia and rigidity with weakness variously combined. The following table roughly shows the relation of the various cord diseases to the physiological division of the cord. Those which are marked by lesions confined principally to given tracts in the spinal cord are denominated system diseases, as distinguished from indiscriminate lesions. As will appear in the consideration of individual diseases, some of these cord lesions, as in locomotor ataxia, are only a part of the morbid findings.

Organic diseases of the cord.	Indiscriminate lesions.			Hemorrhage. Myelitis. Softening. Insular sclerosis. Tumors. Traumatic injury. Compression from bone disease and tumors.
	System diseases.	Lesions of single tracts.	Anterior cornu.	Poliomyelitis acuta. Progressive muscular atrophy.
			Lateral tract.	Primary lateral sclerosis. (Little's disease.) Lathyrism.
			Posterior columns.	Locomotor ataxia. Ergotism.
		Combined lesions.	Anterior cornu and lateral tract.	Amyotrophic lateral sclerosis.
			Lateral and posterior columns.	Ataxic paraplegia. Friedreich's ataxia. Heredocerebellar ataxia. Pellagra. Results of cross lesions.

Lesions of the Cauda Equina.—The descending roots of the lumbar, the sacral, and the coccygeal nerves make up the cauda, which occupies the dural sheath below the upper level of the second lumbar vertebra—a distance of about ten inches. As the roots are given off in lateral pairs, the upper ones are external and soonest pass into the vertebral foramina. Those that are longest, therefore, are situated toward the middle line. It is evident that a lesion involving the dural contents below the conus medullaris will produce a root lesion. This may be partial or complete, and varies according to the level which it occupies and the roots actually involved. The longer roots are usually most affected, even when the lesion is so placed as to embrace all the roots of the cauda extending below the conus. As a consequence, the nerves arising from the lowest cord segments and distributed to the lowest body levels are the ones commonly disturbed. The paralysis, anesthesia, abolition of reflex action, and atrophy that result from a complete root division correspond to the peripheral distribution of the injured nerve roots. These peripheral distribution areas correspond somewhat to the segmental cord areas, but show a marked tendency to follow the distribution of the nerve trunks (Figs. 15 and 16). Both sides of the body are usually affected, but uniform symmetry is the exception while in cord lesions it is the rule.

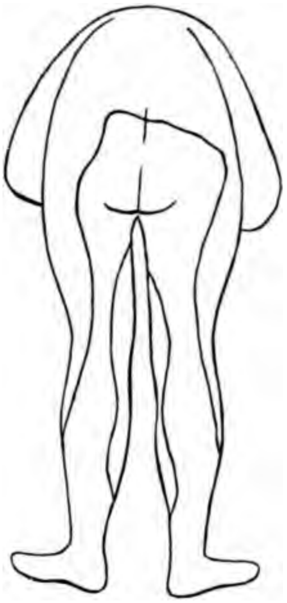


Fig. 129.—Area of anesthesia in a lesion of the cauda, affecting all the sacral roots.

The motor, sensory, trophic, and reflex disturbance that results from a complete division of the caudal roots is the same in character that follows nerve-trunk division. In lesions of less destructive character sensation may exceptionally be but slightly disturbed when motion is quite abolished. Increased reflexes are not encountered. Complete and permanent loss of rectal and bladder reflexes points to involvement of the cord.

In lesions of the cauda, therefore, we have anatomically coextensive sensory, motor, trophic, and reflex symptoms, corresponding to the distribution of the roots making up the sacral and lumbar plexuses or a part of them. The disturbance always affects the lowest portion and ceases at some definite upper level. Most cord lesions, on the other hand, are limited in vertical extent, and the reflex and trophic disorders are confined to the corresponding body segments.

The usual lesions which affect the cauda are vertebral fractures and dislocations, new growths, penetrating wounds, and hemorrhage. In tabes and multiple neuritis, which are sometimes confounded with caudal disease, symptoms are present at higher levels, as in the upper extremities, and in the pupillary reflexes. Injury to the plexuses within the pelvis usually—at least, at first—produces unilateral symptoms.

CHAPTER II.

INDISCRIMINATE CORD LESIONS.

TRAUMATIC LESIONS OF THE CORD SUBSTANCE.

THE spinal cord is sometimes reached by penetrating wounds made by knife, bullet, or other foreign object. It is injured more frequently by the displacement of vertebræ, and this is almost invariably attended by both fracture and dislocation. With the surgical conditions we shall not deal. It is to be noted that vertebral fracture-dislocations are frequently devoid of any external signs of displacement, even when it is found postmortem that the vertebral bodies have been so completely displaced as to actually shear the cord in two. Restitution to a practically normal position may occur at once, either spontaneously or due to the lifting efforts of those who first attend the injured person. These cases all furnish a history of trauma, and usually present local evidence of it. The question that arises regards the location and extent of the lesion in the cord. This must be determined by an application of the considerations set forth in the foregoing chapter.

The prognosis in a case of actual injury to the cord substance is always grave and generally fatal. If the lesion cause complete division, there can be no hope for voluntary control or sensory improvement below its level. Only in very slight injuries from dislocation of the vertebræ or from pressure due to meningeal hemorrhage can much be expected. In such a case complete paralysis may pass away; but when the reflexes are completely abolished in the paraplegic area after the first week, little improvement need be expected. Some degree of disability always persists, and commonly bedsores, cystitis, kidney and pulmonary complications carry off the patient after a lingering helplessness.

HEMORRHAGE INTO THE CORD (HEMATOMYELIA).

Hemorrhage into the spinal cord is not an extremely rare accident. It occurs under a variety of circumstances, and is punctate and multiple or single and more or less extensive. There is also a so-called perforating form simply due to the tendency of the extravasated blood to follow the lines of least resistance in the longitudinal direction of the cord.

Etiology.—Hemorrhage into the cord, like spinal meningeal hemorrhage, with which it is frequently associated, may follow severe spinal concussions and violent wrenchings of the back. Excessive muscular effort, as in lifting, has caused it, and Gowers reports this accident following repeated coitus. A focus of myelitic softening is frequently the seat of hemorrhage, and hemorrhage, in turn, is followed by a zone of myelic softening. It is often difficult to tell which process was the initial one. In some cases of caisson disease, or divers' palsy, hemorrhage has been found due to the too sudden reduction of the high atmospheric pressure under which such work is done. Continued convulsions, as in status epilepticus, intense chorea, tetanus, and asphyxia, may produce punctate hemorrhages and hemorrhagic infiltration, especially in the gray matter of the cord. Purpura and sudden cessation of habitual hemorrhages, such as that from piles and menstruation, may provoke a cord-apoplexy. A dilated central canal and teratological defects and fissures in the cord predispose to it. Changes in the arterial coats are much less active factors than in cerebral hemorrhagic apoplexy, as atheroma and miliary aneurysmal dilatations are of the greatest rarity in the cord. The arterial pressure conditions and the direct cardiac impulse that play so large a part in brain-lesions are also absent.

Morbid Anatomy.—In the punctate and infiltrating variety the cord may merely appear slightly reddened, or small, pinhead clots may be found, particularly in the gray matter. They usually first occupy the perivascular spaces and, aside from traumatic cases, are secondary to myelitic softening. The appearance varies with the size of the extravasation. The large single or focal hemorrhage also finds its usual seat in the gray matter, less commonly in the central canal or in a congenital fissure of the cord. It is usually ovoid in shape, with the long axis vertical, and may perforate the yielding gray substance of the cord for several inches in extreme cases. It causes a fusiform, dark-colored swelling of the cord. The blood usually comes from the larger median arteries or from those entering the anterior horn by the anterior roots, and favors the gray matter as a situation. In rare instances the blood may break through the white columns and even appear to a slight extent in the membranes. Meningeal clots are also commonly present in traumatic cases. Around the clot, after a few days, the cord shows a zone of softening. In cases of long standing the clot may undergo changes similar to those in the brain, and a hemorrhagic cyst remains, with ascending and descending degenerations corresponding to the location of the lesion in the cross-section. A wide area of myelitis, containing a comparatively small and recent clot or hemorrhage into a gliomatous growth, may be encountered as secondary accidents.

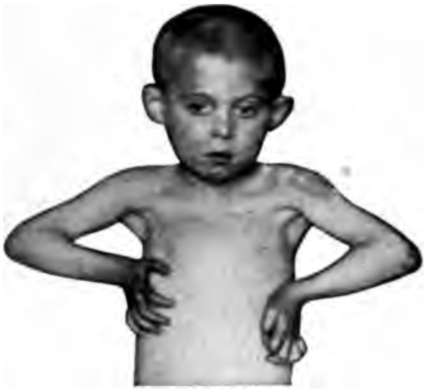


Fig. 130.—Hemorrhage into the gray matter of the cervical cord below the centers for the biceps, and supinator longus; paralysis and atrophy of the triceps and extensors of wrists and fingers.

Symptoms.—A primary focal hemorrhage into the cord is of rapid, usually of sudden *onset*. The infiltration sort, being almost always secondary, may be preceded by sensory and motor symptoms for hours, days, and even weeks. This is also true of the larger clot that forms in a softened myelitic territory, producing sudden exacerbation of the usually precedent paraplegic symptoms. The earliest symptom is commonly one of severe *pains* radiating in the body-segments corresponding to the hemorrhagic focus. These are due, presumably, to the pressure or laceration of the sensory tracts in or near the posterior commissure. Darting pains occur in the limbs, girdling pains in the trunk that may be mistaken for angina pectoris and intestinal or vesical colic. Very shortly—that is, in the course of a few minutes or an hour—after an accident has occurred, and the patient has perhaps walked a short distance, *paraplegic* symptoms appear. These may be partially unilateral at first. The legs weaken, the patient gradually or quickly sinks down, and usually the motor loss is promptly established at its maximum. The condition that now develops depends on the location, extent, and size of the clot, and the amount of pressure it brings to bear on the conduction tracts. Usually there is some improvement, owing to the subsidence of the pressure and of the shock or insult to the adjacent portions of the cord. The development after a day or two of myelitic softening or myelitis, with elevation of the body-heat, usually again emphasizes the paraplegia, and thereafter the course of the disease and its treatment is that of myelitis. The bladder and anal sphincters are commonly immediately relaxed. All the tendon reflexes are diminished at first, but after a week or ten days they begin to increase, and comport themselves as in the spastic state that follows an incomplete cross-lesion of the cord. Early in the attack spasms and tonic convulsions in the muscles supplied by the affected segments and those below the lesion are frequently met with.

The diagnosis is often difficult and it is frequently impossible to exclude a meningeal hemorrhage. Preceding sensory, motor, and temperature disturbances indicate a primary myelitis. Only in those cases where the temperature is normal, the onset abrupt, and the pain of the segmental variety, can a definite diagnosis be ventured.

The prognosis depends upon the location of the lesion. Hemorrhage into the cord in the upper cervical region is almost certainly fatal, and is worse in the cervical and lumbar enlargements than in the dorsal region. The secondary myelitis may extend upward and cause death, or downward and involve the lower levels. Only when sensation improves, motor gain is apparent, and febrile disturbance is passed, is the patient safe. Some lasting local paralysis, wasting, and trophic disturbance result, and more or less permanent spasticity remains. Too often bedsore, cystitis, sepsis, or other complications carry off the patient.

Treatment.—The immediate treatment of the hemorrhage consists of measures to check it. The patient should be placed face downward with the spine elevated as much as possible, and applications of ice or ice-bags made over the length of the cord. Absolute quiet and the determination of blood to the surface, intestinal tract, and extremities should be favored. The arterial tension may be reduced by minim doses of tincture of aconite or veratrum viride every hour, but the use of ergot is not advised. After the first day the treatment is that of myelitis.

THROMBOTIC SOFTENING OF THE CORD.

Thrombosis of the arteries of the spinal cord undoubtedly occurs frequently. The anatomical features of the circulation in the spinal cord render this accident a very likely one. The long course the arterial supply traverses, especially in the lumbar cord, produces a sluggishness of its current that favors the deposition of fibrin if at the same time the arterial wall is nutritionally disturbed. As the spinal arteries after entering the cord are of the terminal variety, it is evident that their obliteration will result in the softening of their irrigation fields. This result has ordinarily been confounded with myelitis and clinically presents the same picture. Gowers denies its occurrence, as does Strümpell, but in syphilitic cases this mechanism is demonstrated by such cases as are described by Williamson[1] and those of Schmaus, Sottas, Déjérine, and Knapp quoted by him. Lloyd[2] also refers to such an one in his own experience. Biernacki[3] reports three cases in full, two of which were syphilitic. Embolism, on the other hand, can practically be excluded, owing to the narrowness of the spinal vessels, the long and tortuous course they pursue, and the slowness of the blood-stream. Experimentally, however, by the injection of inert powders into the circulation of lower animals it has been produced by Lamy,[4] Singer,[5] and others. Though atheroma is infrequent in the spinal circulation, syphilitic cases commonly show endo-arterial and peri-arterial changes. The same are likely to occur in infectious diseases and in many blood states, and favor thrombosis.

The softened area resulting from thrombotic occlusion of the supplying artery, just as in the brain, is likely to become hemorrhagic through the venous back-pressure, and, therefore, may present any degree of hemic discoloration. Into it a neighboring blood-vessel may rupture, with hemorrhagic results.

This condition, as Langdon[6] has well pointed out, comes on rather abruptly without prodromal malaise, fever, or infection. Commonly there is a tendency for the symptoms to increase often by sudden additions and extensions. At first commonly unilateral, the condition tends to become bilateral and the sensory and motor defects symmetrical. The girdle sensation and impairment of the sphincters are less marked and later developed than in acute myelitis, and bedsores less likely to appear. Leukocytosis is wanting. The treatment should be to strengthen the general circulation and use those remedies which favor resolution of local exudates. Most of these cases are syphilitic and require iodid and mercury. In other respects the management and prognosis is the same as that of myelitis.

MYELITIS.

Under the term myelitis a host of spinal lesions have been grouped which have in common the appearance of a local softening, with more or less inflammatory disturbance. Inflammation of the cord-substance is probably never a primary process. Infection may readily reach the cord by the vascular supply. That it does so is evident in the inflammatory lesions of the cord-substance that so often follow the exanthemata and septicemic diseases generally.

The term myelitis is a generic one. In this chapter we are dealing with the indiscriminate lesions of the cord-substance, and, therefore, reserve for separate consideration the variety of myelitis known as acute anterior poliomyelitis that singles out the anterior gray matter, is largely confined to it, and presents a distinct clinical type. We may distinguish a *transverse* myelitis, one that is *disseminated*, and a *central* form depending merely upon the accidental location of the lesion or lesions in the cord. Of these, acute transverse myelitis may be taken as a type, and is the one commonly encountered.

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[1] "Relation of Diseases of the Spinal Cord to the Spinal Blood-vessels," London, 1895.

[2] "Nervous Diseases," by American Authors, Philadelphia, 1895.

[3] "Deut. Zeit. f. Nervenheilk.," Bd. x.

[4] "Archives de Neurologie," 1894.

[5] "Deut. Zeit. f. Heilk.," Bd. xvii, 1897.

[6] "Jour. Nerv. and Mental Dis.," April, 1905.

Etiology.—Acute myelitis may follow *wounds* of the cord, *lacerations* of its substance, from hemorrhage, or from fracture-dislocations of the vertebræ. It has followed violent *muscular efforts*, spinal *concussions*, and falls on the back, but in such cases minute myelitic hemorrhage or other structural lesion may have introduced the program. It has been repeatedly attributed to *cold* and exposure, and this assertion has been handed down so persistently that it seems a permanent fixture in the literature. If cold plays any part, it is, as in pneumonia, to favor the introduction of infection. *Compression* of the cord by disease of the spine or the meninges, or by new growths, causes a localized softening which may girdle the cord. The association of myelitis and *meningitis* is shown in the condition of meningomyelitis already described. The extension of the inflammatory process to the cord is a clearly recognized feature of most meningeal infections. It may follow all the acute *infectious diseases*; probably by an initial thrombosis. *Syphilis* frequently leads to it, and usually by a thrombotic process. It may also result from a gummy tumor or from syphilitic meningitis. These will be considered more at length in the chapter on Syphilis of the Nervous System. Influenza, gonorrhea, all infectious and pyemic conditions, and caisson disease have caused it.

Morbid Anatomy.—On *inspection* an inflamed cord presents a red, swollen appearance and a reduced consistency that may make it pultaceous and even diffluent. The vertical dimensions of the softening vary from one-half an inch to several inches, and usually embrace the full thickness of the cord. Depending upon the amount of extravasated blood and the age of the lesion, the myelitic portion is red, yellowish, or white. It is usually difficult or impossible to distinguish the gray from the white portions of the cross-section, and commonly the softening is so great that the cord breaks down under the slightest handling. All details are then obliterated. *Microscopically*, there is more or less disintegration of the cord-elements. There is usually present an abundance of phagocytic elements, and amyloid bodies are frequently encountered. The axis-cylinders are destroyed, or divided or granular. Sometimes a few of them appear much swollen. The nerve-cells participate in the destruction, and those that are recognizable appear swollen, pigmented, granular, filled with fat-globules, or vacuolated. Their processes early disappear. The vessels are altered, their walls thickened; the perivascular sheaths are dilated with cells, detritus, and hemorrhage. The interstitial tissue is exaggerated in proportion to the duration and intensity of the disease, and in the disseminated form of myelitis forms islets of thickened tissue. There are usually many spider-cells present. The meninges are variably affected by extension of the inflammation to them. In cases of long standing the cord may be reduced to a mere fibrous shred.

If the lesion has been of sufficient duration, ascending degenerations are found in the posterior and direct cerebellar columns, and descending degeneration in the pyramidal tracts. In addition, by the process of contiguous extension the myelitis may propagate itself in either direction from its initial focus along any of the tracts of the cord, or along its gray substance, irrespective of the direction of conduction in the physiological pathways.

In disseminated myelitis small foci of inflammation are scattered throughout considerable portions of the cord, presenting the same minute changes as outlined above. It may require the microscope to detect them, or they may be manifest as small red or hemorrhagic points in the cross-section. In the central form there is cellular infiltration about the central canal, which is often dilated and choked. The nerve-roots arising from the focus of inflammation show neuritic changes and degeneration, with corresponding muscle changes. Should a myelitis be infectious from the first, or subsequently infected by pus-producing bacteria, abscess formation results. From such a cord-abscess the meninges may become infected and a purulent meningitis ensue.

Symptoms.—The symptoms of myelitis are as diverse as the cases, and each case varies with the vertical or transverse location of the lesion or lesions, with their number, extension, severity, and character. The *onset* is modified by the initial cause of the disease in the cord. When hemorrhage is the first step, it is apoplectic in suddenness. Traumatism has its own history. The infectious diseases have their individual clinical features upon which the myelitis is grafted. In less well-marked antecedent states the onset of the paraplegic features of the disease may be unheralded by any subjective or objective phenomena. A few days of malaise, of slight fever, or of fleeting paresthesia may indicate the systemic condition which eventuates in myelitis. In other but rare instances convulsions, high temperature, and rigors declare the toxic process and usher in the spinal symptoms. These consist usually at first of intense *pains*, which may be darting in character, extending along the limbs or girdling the trunk. There is more or less tingling and numbness. The distribution of such sensory disturbance in relation to the cord-segments should be significant. Shortly after—in a few minutes in hemorrhagic cases, in a few hours or a few days in infectious forms—more or less *paraplegic weakness* is developed, which involves everything below the segmental location of the disease. The motor loss may be sudden or gradual, complete or partial, but usually is insidious, progressive, and does not reach an absolute degree. The control of *bladder and bowels* is usually disturbed early, with more or less incontinence or retention. In some instances the motor features come on and progress with the sensory disturbances, or even precede them. The order of symptoms depends upon the portion of the cord first and most diseased and the destructive or irritative character of the lesions. It follows that spasmodic twitching of the limbs may occur, but, as a rule, there is complete flaccidity at first.

The number and variability of the symptoms are so great that they can best be presented under several heads.

Sensation.—The upper level of the sensory disturbance is usually marked by a hypersensitive band corresponding to the upper segmental extent of the cord-lesion, and due to its irritant action. This is always present in cross-myelitis, and should be diligently sought, as it is of the greatest localizing importance. In the entire area below the hypersensitive zone sensation is more or less blunted and may be completely lost in all its modes. When the cross-lesion is less complete, sensation or motion, or both, may be only partially involved. When the lesion is practically central, we have the peculiar dissociation of touch-sensations that marks lesions in this location. There is analgesia and loss of temperature sense, with preservation of tactile perceptions. Involvement of the posterior roots and extension of the inflammation to the meninges are marked by local pain and tenderness over the spine at the level of the lesion and above it. The girdle pain has the same topographical significance as the hypersensitive zone, and usually corresponds to it. The patient often complains of paresthetic sensations below the lesions, even in completely anesthetic territory, or in the abdominal viscera. These may be misleading to both patient and physician. The greatest care must be exercised in testing the cutaneous and other sense perceptions, as indicated in Part I. The sensory symptoms frequently vary greatly during an attack unless the lesion completely severs the cord. The reappearance of sensation where previously wanting is a good sign, just as the increase of sensory loss is indicative of extension of the disease and of bad import, sometimes of fatal significance if toward the upper cervical levels.

Motion.—The loss of motion corresponds to the same segmental distribution as the anesthesia, and may be complete or partial. The anterior gray matter in the inflammatory focus is usually completely destroyed, or at least its functions are completely inhibited for the time. The muscles controlled by this portion of the cord are paralyzed. The amount and degree of paralysis below the lesion depend upon the completeness of the cross-lesion, and varies within wide limits. In the disseminated and multiple forms of inflammation various functionally related groups of muscles may be singled out, as in acute poliomyelitis. Here, also, the motor loss may show any degree of incompleteness. The loss of sphincter control is usually present from the first and persistent. When the lumbar cord is affected, incontinence of urine and distention of the bladder and bowels follow the destruction of their spinal centers. The distribution of the paralysis is, therefore, always dependent upon the segment or segments of the cord that are diseased, and has a common tendency to paraplegic distribution, involving both sides more or less symmetrically. The distribution of symptoms in rare instances suggests the Brown-Séquard syndrome, due to a lateral hemicordal division (see p. 55).

Reflexes.—The reflexes whose spinal centers are situated in the inflammatory focus are abolished. Below that level they are usually diminished at first, but at the end of a week or ten days commence to increase in vigor and gradually attain extreme exaggeration. Should the lesion actually divide the cord, they are abolished, as in traumatic cases, but the preservation of a very few fibers in any portion of the cord's cross-section seems sufficient to allow of their exaltation and the development of spasticity and rigidity. All forms of clonus and intensification of reflex activity may be encountered. The tendency is for the lower limbs to be rigidly extended, adducted, and sometimes crossed. Less frequently flexion predominates and the legs are flexed upon the thighs, which are firmly applied along the anterior surface of the trunk. The clonic condition of the limbs sometimes serves to jerk them about sharply upon slight skin friction, even by the removal of the bedding or any gentle manipulation. The flaccidity of the early days is replaced by a hypertonicity, so that the muscle-masses may stand out prominently. Contractures may result and often do. Priapism is commonly present in cervical myelitis, and frequent in inflammation of the dorsal cord, but absent in lumbar involvement. Lesions in the cervical region are commonly attended by a dilated pupil, but in some cases, especially of the disseminate variety, optic neuritis is present, and pupillary responses are variously modified. The condition of the vesical and rectal reflexes is one that should early engage attention. If the lesion involves the lumbar cord, the sphincters are usually relaxed and incontinence follows, but there is a tendency to urinary retention through relaxation of the visceral walls, and cystitis is easily established. Again, when the lesion is above the lumbar cord the sphincters operate automatically, and both feces and urine are discharged at intervals; but, again, the bladder-wall is likely to yield, or acts ineffectually without the aid of the abdominal muscles; urine is retained, becomes ammoniacal, and, through contamination by catheter or otherwise, cystitis develops. Rupture of the bladder through distention and ulceration, causing peritonitis, has been seen. Some cases present a most obstinate and excessive gaseous intestinal distention. This occurs most frequently when the lesion is above the mid-dorsal region.

Trophic Changes.—The muscles that are anatomically related to the diseased cord-segment waste promptly and show the reaction of degeneration. In addition the inert limbs lose in size from disuse and considerable emaciation is frequently presented, but the electrical changes are lacking and the reflexes are usually increased. The general vasomotor and trophic conditions below the lesion are disturbed. A slight stroke of the finger-nail upon the skin will usually present a line of persisting vascular stasis like the meningeal tache, and at first the paralyzed portions show an elevation of temperature even above that in the mouth. In cases of long standing the local temperature is abased and the paralytic members are blue, cold, cyanotic, and often edematous. The skin is often dry, harsh, and scaly, and readily breaks down under pressure, forming ugly, sluggish, unmanageable bedsores. The tendency to bedsore is prominent from the first, and it is in these cases that the sacrum is sometimes denuded within a few days under the continued influence of pressure, irritation from urine or feces, and the dystrophic element. The entire vesical mucous lining may exfoliate from disturbed trophic conditions. Joint-lesions of the arthropathic sort are rarely induced.

The general nutrition of the patient suffers to some degree, but less than would be expected, and it can usually be maintained at a reasonable level. The danger arises from extension of the myelitis and from complications arising through cystitis, bedsore, nephritis, and septicemia, or concurrent acute infections, such as pneumonia.

Course.—Acute cases reach their maximum in a few days, others in a few weeks, and then, if death does not result, a long stationary period or one of gradual improvement or decline succeeds. The occurrence of an extension of the inflammation may, at any time, jeopardize life by invading the respiratory apparatus. Acute bedsore is always a dangerous complication, and cystitis is hardly less so. When spastic features develop, they rarely recede to any considerable extent, and imply permanent disability and the paraplegic state. Sensation or motion may return singly. The localized wasting due to involvement of the anterior gray never repairs, and adjacent portions of the gray matter may subsequently be involved. Death may take place early from cardiac and respiratory failure or follow at any period from exhaustion due to the primary infection or that secondary to bedsore, cystitis, nephritis, septicemia, or is caused by a gradual extinction of the vital energy. The cases that recover bear the indelible marks of the disease in weakened and spastic legs, areas of anesthesia, sphincteric paresis, and local atrophies variously distributed in accordance with the seat, extent, and intensity of the cord-injury. These furnish cases of so-called chronic myelitis, but inflammation has subsided and the conditions presented are due to the degenerations that follow the primary lesion. They are more fully described under the head of The Paraplegic State.

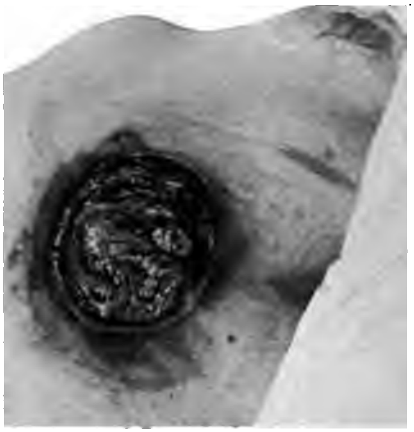


Fig. 131.—Dystrophic bedsores over trochanters and sacrum in a case of transverse myelitis.

Diagnosis in myelitis presents numerous problems and requires painstaking examination and study. We have to ask ourselves: (1) Whether the cord is actually diseased; (2) the extent of that disease—namely, its localization—and (3) its origin. Unless there is loss of certain groups of cord-functions anatomically related, we can not incriminate the cord. Of these, the most important are early loss of motion of a paraplegic distribution and relaxation of the sphincters. Corresponding sensory disturbance or anesthesia surmounted by a band of hyperesthesia will almost surely be added to, if it does not precede, the motor loss. The onset is usually acute, and in the hemorrhagic variety it is sudden. Extreme pain of a radiating, segmental variety and sudden onset suggest an initial vascular lesion. After ten days we may detect muscular atrophy corresponding to the lesion and usually increased reflexes below the lesion, which usher in the spastic stage that is to persist as a paraplegic state if the patient survives. Even before this time the loss of faradic response in the muscles innervated by the spinal gray embraced in the inflammatory focus may indicate the myelitis and its vertical extent. To the further localization of the cord disease we bring to bear the considerations set forth in the preceding chapter. Our main early guide is loss of reflexes and the vertical extent of such hiatus in the chain of spinal reflexes.

In *poliomyelitis* sensory deficiency is not present and the motor loss is usually of monoplegic outline. In *meningitis* we have the early and persistent root pain, tenderness over the spine, and retention of motion and reflexes. In *Landry's palsy*, or acute ascending myelitis, we meet the steady advance of the paraplegia from the feet and legs upward, with undisturbed sensation, faradic activity, sphincteric control, and tendon reflexes. In *multiple neuritis* the slow onset, involvement of all four extremities, major affection of the extensors, and paresthesiæ at the distal ends of the limbs are significant. In *hysteria* the sensory disturbance has a characteristic outline, the reflexes are not greatly disturbed, trophic changes are not present, and hysterical stigmata are obtainable. The mistake usually made is to overlook an organic disease because hysteria is also present.

The diagnosis of myelitis having been reached, it is always in order to question its origin. This may be evident from the history or presence of traumatism, acute infectious disease, septicemia, syphilis, or other cachectic state.

Prognosis in such a generic condition as myelitis must be based upon general rules applied to the individual case. The outlook is always grave as to life and positively bad as to complete recovery. If the patient is not carried off during the first few days by the implication of the cardiorespiratory apparatus, or by the overwhelming systemic effect of the infection, which is perhaps only locally manifest in the cord, and if he reaches the end of the first week without any indications of extension of the myelitis, he may be considered out of immediate danger. If at the end of three or four weeks he does not show at least some slight return of motion and sensation, it is not likely that these will ever greatly improve. On the other hand, distinct improvement within the first two or three weeks is usually followed by rapidly progressive gain almost to the point of entire recovery. When spasticity appears it implies descending degeneration in the pyramidal tracts and lasting disability. Cervical myelitis is almost invariably fatal. Involvement of the dorsal cord is much less disastrous than when the lesion invades the lumbar enlargement and perforce permanently cripples the sphincter control and the legs. Acute bedsore and acute cystitis are ominous complications. Local muscular atrophies are subject to this rule: If faradic response can not be obtained in such muscles at the end of the first two weeks, they may be considered as permanently impaired. In syphilitic cases that reach a marked degree of paralysis we may hope to prevent extension of the disease and sometimes to secure a marked recession of the paresis but never for complete cure.

Treatment divides itself into three parts: (1) That of the causal condition; (2) that of the invasion stage, and (3) that of the paraplegic state. Traumatic conditions, pressure from meningeal hemorrhage or new growths require surgical measures. Any systemic infection or infection atrium must be directly dealt with. For the local condition the treatment outlined for meningitis is available. During the invasion period the inflamed cord should be kept elevated by placing the patient in the prone posture. Local applications of ice are useful. Active cathartics should be used if the patient's strength warrant their administration. Quiet, and above all cleanliness, must be secured and local pressure avoided. Early attention to the bladder is usually needed but catheterization is only indicated when vesical retention can be determined by palpation and percussion, and then must be carried out with the most scrupulous antiseptic precautions. Febrile disturbance is usually a part of the original causal process and to be met accordingly.

After the first two or three days, if the case runs on, a water or air bed will be found of the greatest service in distributing pressure. The heels, elbows, and other bony prominences in the paralytic field should be carefully padded with cotton. The constant use of a bed urinal will often help to keep the patient dry. Alimentation and supporting treatment will require thoughtful attention. At the end of a week careful massage and faradization may be employed, if not contraindicated by surgical conditions in the spine. Gentle frictions and kneadings of the muscles are indeed advisable from the first day, and the position of the paralyzed limbs should be changed hourly if only slightly, as is automatically done in healthy sleep. When a bedsore develops, its best management depends upon keeping it perfectly dry. To this end a soft gauze pad and an abundance of pulverized boric acid should be used, but the dressing must not be cumbersome or so arranged as to exercise pressure and the patient's position must also conform to this requirement. Cystitis requires careful, thorough, skilful catheterization and washing out of the bladder every eight or twelve hours. The administration of salol, beta-naphtol, or urotropin also tends to render the urine unirritating and to some extent disinfects the bowel contents. The management of the case now resolves itself into one of good nursing and attention to obviate complications, especially malposition of joints and contracture deformities.

As motion and power reappear the patient should be encouraged to use the limbs intelligently. By concentrating his attention upon a certain movement he can often produce it after a number of attempts.

Local nutrition must be kept up by massage and electricity. The use of the faradic brush to the anesthetic area is of service in restoring sensation. The general physical condition and usual constipation require constant attention.

THE PARAPLEGIC STATE.

The paraplegic state is the usual termination of a myelitis or any indiscriminate cord lesion that does not end fatally, and corresponds in some sense to the "hemiplegic state" that supervenes upon brain-lesions. Very often arising from such cause as acute myelitis, it is mistaken for a chronic inflammatory condition and denominated chronic myelitis or confounded with ataxic paraplegia and even with locomotor ataxia. The descending degeneration in the pyramidal tracts is a consequence of any lesion which involves the upper motor neuron. When due to bilateral lesions above the cord it is best called a diplegia. Cross-lesions of the cervical cord are usually promptly fatal, so that the paraplegic state arising from indiscriminate lesions only reaches its later and characteristic development in cord lesions below that level.

Etiology.—All the indiscriminate cord lesions, such as traumatism, hemorrhage, thrombotic softening, myelitis, tumors, and pressure from meningeal and spinal diseases and growths, give rise to a paraplegia which is more or less pronounced in proportion to the extent of the lesion. In addition, the so-called system lesions which are marked by sclerotic degeneration of the lateral tracts produce paraplegic conditions, but without distinct anesthesia. We find it also in Little's disease, ataxic paraplegia, family cerebellar ataxia, and amyotrophic lateral sclerosis. The only common fact is the degeneration of the motor tracts in the lateral columns. Diseases of the upper neuron arising from intracranial lesions have been considered in connection with diseases of the brain (Part III).

Symptoms.—The symptoms of this secondary state vary greatly and are dependent upon the nature, extent, and activity of the initial lesion. In the system diseases the spastic paraplegia, as in Little's disease, may be really a diplegia from embryonic defect, and congenitally present. In the family ataxias the defect is also teratological, but the symptomatic development is postnatal and usually insidious. Ataxic paraplegia and amyotrophic lateral sclerosis also develop very gradually and usually in adult life. They all in common possess defects of the pyramidal tracts marked by loss of muscular control and strength especially developed in the lower extremities, and by increased reflexes and spasticity. The special paraplegic features which they possess will be taken up in the description of the various system diseases. In this present connection the paraplegic state arising from indiscriminate lesions is principally in view.

If the initial cord lesions be acute, as in traumatism, hemorrhage, and myelitis, the motor loss is promptly established and the muscles are flaccid and inert. The sphincters are usually relaxed. The reflexes represented in the diseased cord segments are abolished and those below the injured focus are diminished. If the cord is entirely severed, they are completely and permanently lost and rigidity does not ensue. When the cord is not entirely divided, at the end of a week in some cases, in others several weeks later, the increasing reflexes indicate degenerating lateral motor tracts and usher in the spastic features that are to permanently remain.

When the inciting cause of the paraplegia is of gradual development, as in pressure conditions arising from Pott's disease and spinal neoplasms or new growths in the membranes or cord, the clinical program is very different. Root pains, girdling the trunk or streaking into the limbs, depending on the segment location of the lesion, are early symptoms and usually there is complaint of heaviness, weakness, and clumsiness in the legs. This increases either steadily or by stages, in the intervals of which some amelioration may occur, and eventually the hypersensitive girdle and subjacent anesthesia are more or less well developed. Sphincteric loss is a late feature and is usually preceded by months of slighter degrees of incontinence. As the pressure increases, compression of the pyramidal tracts gradually develops; they degenerate and the reflex activity is correspondingly exalted. It is in these cases of slow cord compression that the spasticity, reflex automatism, and muscular rigidity with resultant contractures reach their highest exemplification.

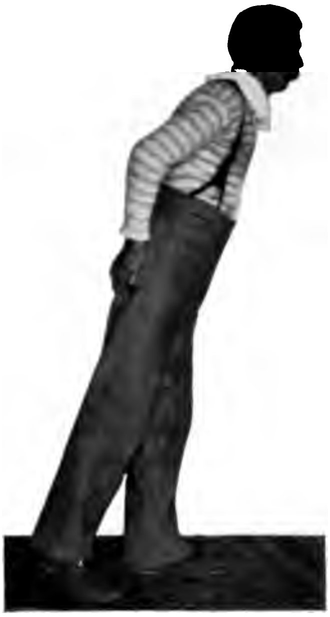
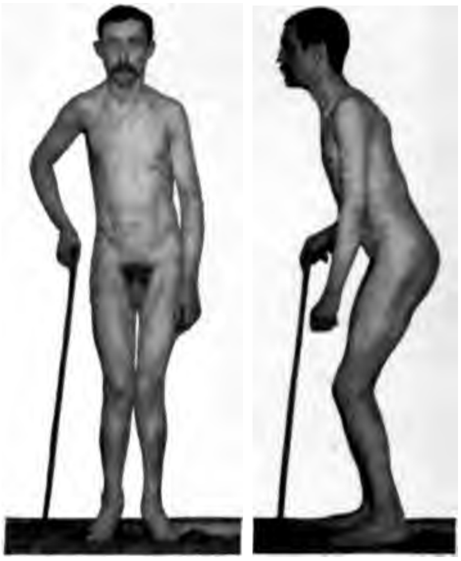


Fig. 132.—Paraplegic gait.



Figs. 133 and 134.—Station in spastic paraplegia due to syphilitic myelitis, showing rigidities, flexed knees, and adducted thighs.

When the acute cases have improved enough to walk or the insidious cases have attained a fair degree of development, the *gait* is highly characteristic. The patient's feet once planted seem glued to the floor, and the upper portion of the body is inclined forward in advancing. The pelvis is elevated on one side and that limb is then brought or dragged to a position under the center of gravity like a rigid pendulum (Fig. 132). It may even be aided by the hand. As it advances it is shaken by clonic movements, and when planted these may cause it to execute several dancing steps before it is securely placed, during which the heel is forcibly elevated and the patient may be bodily jostled up and down. The body is then again inclined forward over the supporting limb, and the opposite member is in turn carried a little in advance of its fellow. These short, jerky, halting, clonically disturbed steps are frequently rendered more difficult by the over-action of the adductors of the thighs which displace the limbs inward, cause the knees to interfere with each other, and sometimes even induce a cross-legged gait. Progress, in spite of the disturbance of the gait, is usually made in a fairly straight line, unless some slight obstacle over which the patient is sure to stumble, or an unusual amount of clonus, cause him to swerve. In nearly every respect the gait is the opposite of the steppage of multiple neuritis.

If walking is impossible, the attitude in standing may still indicate the spastic state. The tendency to contracture usually draws the knees forcibly together and partially flexes the knee- and hip-joints (Figs. 133 and 134). A sudden reflex contraction of the calf-muscles may cause the patient to rise on his toes or throw him to the ground. The knees often suddenly give way.

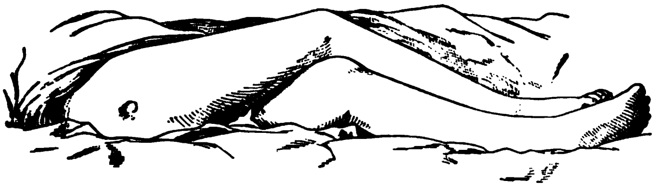


Fig. 135.—Paraplegia from spinal fracture. Attitude in bed. Thighs adducted and crossed. Bedsore over trochanter.

When the patient is bedridden and the paraplegic state is developed to an extreme degree, the lower limbs may be rigidly extended on the pelvis and at all their joints. Adduction is strongly marked and may even cross the limbs. If one foot be lifted from the bed, the rigidity may serve to cause the other one to follow, as if the hip- and knee-joints were ankylosed. In other severe cases flexion predominates, and the lower extremities are rigidly folded upon themselves and upon the trunk, so that the heels are drawn up to the buttocks, the knees to the sternum.

The reflexes are always exaggerated after the early flaccidity of acute cases has receded, and in the later stages become excessive to an incredible degree. The slightest tap on the patellar tendon forcibly throws out the leg, and the whole extremity may be seized with a clonic activity that provokes jerky, more or less rhythmical, movements which may also appear in the opposite limb, and finally end by a jack-knife contraction that violently flexes both extremities upon the trunk and at the knees. These reflex storms may even follow the slightest cutaneous impression, such as gently removing the bed-clothing, and are not infrequently the source of much pain and suffering. The vesical and rectal reflexes usually work automatically in protracted cases of paraplegia, and are only slightly under the patient's control, or entirely beyond it. Lesions in the lumbar cord may destroy them. When the upper dorsal or lower cervical cord is affected, priapism is frequently present, and cervical lesions cause dilatation of the pupils.

All of these conditions vary from the slightest to the most extensive degree in different cases. Slow compression of the cord, as by tumor, gives the most highly colored picture, ataxic paraplegia perhaps the least.

The amount and character of the sensory disturbance naturally depend on the extent of damage to the posterior half of the cord. Atrophy of muscles is in the same way dependent upon the destruction of the anterior gray and is related to the vertical extent of that destruction. More or less general emaciation, due to inactivity and depressed systemic conditions, is commonly present. Contractures produce deformities that still further cripple the patient.

The prognosis naturally depends on the character of the cord lesion. The removal of pressure in Pott's disease and in spinal tumors by operation, frequently results satisfactorily and the cord functions return to a normal state. When the spasticity is the result of destructive lesions of the cord, as in myelitis and traumatism, some disability is bound to persist. It may eventually be very slight or it may progressively increase. Every case must be individually considered.

Treatment.—The treatment of the paraplegic state consists of: (1) Removing the cause if possible; (2) preventing contracture distortions or correcting them by surgical means, such as tenotomies and orthopedic apparatus; (3) the use of electricity and massage to atrophied muscles; (4) general measures to build up the systemic condition; (5) local applications over the spine to control any lingering inflammatory state or to hasten absorption of exudate, and (6) of the administration of spinal sedatives to reduce the reflex excess. Local conditions, such as indolent ulcers, cystitis, and incontinence, furnish their own indications. It will often be found that massage and electricity produce so much reflex stimulation that they must be discontinued. Hot baths often produce a grateful relaxation. Counterirritation to the spine and the resources of hydrotherapy sometimes are of great advantage.

LANDRY'S PARALYSIS (ACUTE ASCENDING PARALYSIS).

The case observed by Landry,[1] reported in 1859, presented these striking peculiarities: An acute paralysis beginning in the legs, extending to the trunk and arms, soon involving the bulbar centers, and terminating fatally. The paralysis was not marked by loss of sensation; the sphincters were not involved. The muscles retained their faradic irritability, the mind was not disturbed, and the temperature was practically normal. No changes were found in the central nervous apparatus by microscopical examination, but the spleen was acutely enlarged.

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[1] "Gazette Heb.," 1859.

Since that time several hundred cases have been reported as instances of Landry's paralysis, some of which correspond closely to, while others depart materially from the original outline. Of these latter we may say that most of them were cases of multiple neuritis, some were cases of poliomyelitis anterior, and others were cases of cross or disseminate myelitis with extension. With improved methods of investigation there has gradually accumulated a considerable number of cases which are clinically true to the prototype, but show organic disease in the spinal, bulbar, and even in the cerebral matter and in the nerve-roots and peripheral nerves. With the development of bacteriology we may now add typical cases in which infectious bacteria have been observed in the spinal cord or obtained by cultures from it. We are, therefore, justified in defining Landry's paralysis as an acute infectious ascending paralysis due to an infectious or toxic condition that may induce a myelitis largely confined to the anterior gray matter of the cord and which may, in addition, cause root involvement, peripheral neuritis, and changes in the medulla and cortex of a similar nature to those in the cord.

Etiology.—This form of spinal palsy is about four times as frequent in adults as in children, according to the tabulation of selected cases by Bailey and Ewing,[1] and affects males nearly three times as often as females. It has followed close upon or occurred during attacks of numerous infectious diseases and conditions. Small-pox, tuberculosis, typhoid, pneumonia, diphtheria, syphilis, influenza, pelvic cellulitis, the puerperium and obscure febrile disturbances have seemed to play a part in its inception. Alcoholism [2] and exposure to cold are also rather frequently mentioned, but in numerous cases not the slightest cause has been detected and the patient apparently was in good health previous to the paralysis. The toxic features of many of the alleged causes jump with the clinical manifestations which are highly significant of an infectious state, and coincide with the few bacteriological findings that have been reported. The idea of a toxemia which has an elective action for the spinal gray producing first dynamic conditions, later followed by histological changes, best explains the various postmortem findings and the clinical manifestations of the disease. E. F. Buzzard [3] isolated a micrococcus in pure culture from the blood of a case of Landry's paralysis and apparently the same organism was found in large numbers in the dura mater of the same patient. Subdural injections of the cultivated coccus produced rapidly spreading paralysis in a rabbit and the organism was again obtained from the blood and dura. The changes in the nervous system of the patient and rabbit were of the kind produced by toxins and in neither could the microbe be demonstrated in the nervous structures or even in the pia arachnoid. That the infection or toxin reaches the cord through the blood is abundantly shown by the perivascular changes, and the frequent limitations of the myelitic invasion to the territory irrigated by the anterior median arteries and the vessels reaching the anterior horns along the motor roots.

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[1] "N. Y. Med. Jour.," July, 1896.

[2] Krewer, "Zeitschr. f. klin. Med.," Bd. xxxii.

[3] "Brain," Spring, 1903.

Morbid Anatomy.—In the older reports, as in the first instance, no morbid changes were detected anywhere in the nervous system. As methods improved and data multiplied, reports of myelitic softening, especially in the gray matter, but also implicating the neighboring white fibers of the dorsal and cervical cord, degeneration in root-fibers and peripheral nerves, and, finally, changes in the medulla, cerebrum, and cerebellum were made. A majority of cases, whether showing cord changes or not, presented an acutely enlarged and softened spleen and often engorged lymphatic glands. Of late no case that has been systematically and competently examined has given negative findings. The cellular structures of the anterior gray or the cylinder processes arising in the motor cornual cells are found disturbed. Eisenlohr, Ross, Hoffman, Immerman, Curschmann, Ketli, Hlava, Marinesco, Bailey and Ewing all found such changes varying from fragmentation of the cylinder process or slight swelling of the cell bodies and chromophilic changes of the cell protoplasm to well-defined poliomyelitis and diffuse cellular infiltration throughout the spinal gray. Frequently the blood-vessels show a perivascular small-cell infiltration and the motor cells present marked degenerative changes. In cases of sufficient intensity and duration the peripheral nerves are degenerated and muscular degeneration and even atrophy are added. The cranial nerve nuclei and the cellular and vascular elements of the cerebral and cerebellar cortex were similarly affected in Ewing's case.[1]

Bacteriological examinations have sometimes been made with negative results and suitable stains have often failed to show bacteria in the cord sections. Remlinger [2] met with this experience even when cultures from the cord readily developed streptococcus pyogenes and streptococci were also found in the lymph-spaces of the gray matter of certain other portions of the cord. Eisenlohr found the staphylococcus pyogenes aureus in all cultures and staphylococcus aureus in cultures from the spleen. Centanni found a rounded bacillus in the peripheral nerves. Giuzetti found chromogenic bacilli in cord cultures. Marinesco found cocci in the ganglion-cells. Roger and Josné [3] have demonstrated the pneumococcus. Thoinot and Masseline have produced spinal paralysis in rabbits by the intravenous injection of staphylococcus pyogenes aureus and of bacillus coli.

Symptoms.—Acute ascending paralysis may develop during an attack of some infectious disease or may follow it. In several epidemics of poliomyelitis such cases have been encountered. Frequently, however, it comes on without malaise, fever, or premonitory symptoms, usually without tingling, numbness, or other sensory disturbance. A feeling of weakness begins in the feet and legs, and slowly creeps upward, becoming more and more pronounced in the lower levels as the disease mounts. It may affect one leg first or most. At the end of two or three days or a week the lower extremities are completely paralyzed and the weakness has involved the trunk and upper limbs. The breathing becomes superficial from involvement of the thorax, and difficulty in swallowing soon appears. In severe cases every voluntary muscle below the face is completely paralyzed and relaxed, and even the cranial nerves may be involved, especially the oculomotor, facial, and hypoglossal. Cerebral and mental symptoms are absent until the dyspnea or cardiac failure is pronounced and induces them. The sphincters are not, as a rule, relaxed; there is no tendency to bedsores or dystrophy; the tendon and superficial reflexes are usually present; the electrical responses are normal; and sensation, together with the special senses, is not perverted. If a fatal issue do not occur, the symptoms of paralysis slowly recede in the reverse order of their appearance, and when they have distinctly subsided from the upper levels recovery may be anticipated.

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[1] *Loc. cit.*

[2] "Comptes Rendus de la Soc. de Biol.," April, 1896.

[3] "Presse Méd.," July 27, 1898.

In some cases the onset is reversed, the upper extremities first showing weakness; and, indeed, the ordinary type may be greatly modified, as can be readily understood from the varying anatomical distribution of the organic lesions in well-authenticated observations. In two cases falling under the writer's attention, the clinical history was typical, complete wasting of isolated muscle-groups in all four extremities occurred, and persisted for years, without any appearance of ultimate improvement. Paresthesia and dysesthesia are not rare, and anesthesia may gradually follow the paralytic invasion, advancing in a similar manner. The reflexes may also subside and disappear in an ascending progression. Even electrical modifications and the reaction of degeneration are encountered. The progress of the paralysis may stop at any point, and then recede. A temperature of 101° to 103° F. has been observed, but, as a rule, it does not rise above the normal. Profuse perspiration sometimes and splenic enlargement frequently are encountered and bespeak the toxic state.

Course.—The course from inception to fatal termination may be very brief,—less than two days,—and fatal cases usually end within ten days. Prolonged cases may only reach their acme in a month. After a stationary period of varying length in the hopeful cases, improvement takes place usually in a retreating order, but convalescence is slow and may require months. On the other hand, it may be rapid, or, as in the cases previously mentioned, permanent injury may result.

Diagnosis.—The diagnosis in some cases must necessarily be extremely difficult, but in the typical form is readily made, providing the existence of this rare disease is kept in mind. It rests upon the method of invasion, the pure motor paralysis, the comparatively negative conditions as to reflexes, sensation, and electrical reactions, and the history of some possible toxemic state. Somes cases are complicated by hysteria, which is capable of greatly obscuring the diagnosis. When slight electrical changes and paresthesiæ are present, it is impossible to exclude neuritis, and the occurrence of peripheral nerve-lesions in some instances has already been pointed out. In meningitis the pain and rigidity are distinctive. In cross-myelitis we have all spinal cord functions involved below a definite level and lack the ascending features.

Prognosis is always grave, since even in the irregular and prolonged cases one can not foretell at what moment bulbar symptoms may appear, and the main danger to life depends on their presence. Rapidly ascending symptoms imply a speedy termination, but there is no invariable rule. Only when the tide has turned and symptoms are receding can one entertain a reasonably hopeful prognosis. The presence of neuritic conditions or of electrical changes implies a prolonged convalescence and doubt as to ultimate complete recovery. Where cerebral symptoms appear they are of bad import, signifying either profound toxic conditions or the near approach of death from cardiac or respiratory failure.

Treatment will be directed against any general toxic condition present or reasonably suspected. The salicylates, tincture of the chlorid of iron in full doses, bichlorid of mercury to the point of toleration, thorough cleansing and disinfection of the alimentary tract, supportive diet, conservation of nervous energy and strength, are valuable. To the spine a narrow sinapism the whole length of the back, frequently repeated, is of service; even the thermocautery is advised by some. The paralyzed limbs should be gently massaged to improve circulation and give comfort. When swallowing becomes difficult or impossible, feeding by the stomach, nasal, or rectal tube must be adopted, and the preference is for the nasal tube, providing care be exercised to avoid passing it into the larynx. During convalescence, massage, electricity, local douches, tonics, generous diet, and general measures are the main reliance.

CAISSON DISEASE, OR DIVERS' PALSY.

Workmen and others subjected to high atmospheric pressure, as in descending to great depths in diving apparatus, or in making certain excavations by caisson construction, are frequently affected with cerebral symptoms and paralytic conditions of mainly a paraplegic character. The disturbance varies in intensity from slight giddiness and neuralgic pains to paraplegia. Even sudden death may occur. The symptoms appear while the air-pressure is being reduced, or within the following half-hour. In a minor degree, high altitudes, as in mountain climbing and ballooning, furnish analogous conditions and symptoms.

Etiology.—The cause of divers' palsy is not so much the increased atmospheric pressure, as its sudden reduction. A number of predisposing causes have also been fairly determined. Advanced age, alcoholism, heart and kidney disease, obesity, hunger, and any condition of physical depression furnish a liability to its onset. On the other hand, those who have for months been gradually subjected to increasing air-pressure acquire in some degree an immunity by habituation. The length of exposure and the amount of pressure are followed by proportionate effects, but symptoms rarely result unless the pressure reach two atmospheres—thirty pounds.

The mechanism producing the palsy or the slighter symptoms is furnished by the vascular apparatus. Various theories have been advanced to explain the results. One supposes that during the time of increased air-pressure the superficial and peripheral parts of the body are exsanguinated and the central organs actively congested to a similar degree. This congestion reaches such a point that the capillary field becomes paretic from distention, and can not promptly deliver itself of its superabundant blood when pressure is removed from the periphery. A passive congestion then ensues, with a stagnant blood-current. The lower cord is placed at an especial disadvantage through its arterial arrangement, which mechanically conduces to maintain the vascular stagnation. Serous effusion into the cord and meninges may thus follow, or the effusion may be hemorrhagic.[1]

Another and apparently more important feature is added to the congestive state. The oxygen, nitrogen, and carbon dioxid of the blood, increased under the high pressure, are liberated by restored low pressure, and, expanding, fill the passively congested vessels with gaseous bubbles, producing something like an air embolism. The gases also escape into the tissues. Catsaras, of Athens, has seen gas bubbles in the vessels and even in the cord parenchyma of dogs subjected to high air-pressures, and gas has been seen in the tissues and vessels in some human autopsies after death from this cause. If vascular accidents do not follow, the circulation is gradually equalized, the oxygen is absorbed or removed by respiration, and symptoms subside. P. Bert, Hoche,[2] and Snell [3] have practically proven the gas theory. Edemata, effusions, and hemorrhages, naturally, produce more or less lasting symptoms in the brain and especially in the cord.

Morbid Anatomy.—In the few autopsies on record, which have all been made some days or weeks after the onset of the attack, the cord has always been found abnormally congested. Small hemorrhages have been seen and diffuse myelitis with degenerative tracts have been noted. In some cases there has been an edematous condition of the membranes and cord. Hemorrhages into parenchymatous organs and mucous surfaces have also been observed. The secondary myelitis is most pronounced in the dorsal half of the lumbar cord and the anterior horns are practically unaffected. In other words, that portion of the cord which has the best vascular supply, and where stasis and edema would first subside, ordinarily escape injury. The painful manifestations of the attack are perhaps explained by the location of the vascular disturbance in the sensory portion of the cord.

Symptoms arise as the air-pressure is being reduced, or shortly thereafter, and may appear while the men are in the locks or chambers that are placed between the various pressures or after the patient has gone some distance in the open air. At first the cerebral features predominate; headache, giddiness, faintness, nausea, vomiting, delirium, double vision, and even coma may precede or accompany the spinal symptoms. These consist of paroxysmal pains, frequently of great intensity. They usually are felt in the legs, but may affect the trunk or upper limbs. Their relation to the posterior portion of the cord has already been mentioned. Soon the patient feels numbness and weakness in his legs, which may increase rapidly to complete paraplegia, usually confined to the lower extremities, but it may extend downward from any spinal level. It involves motion, sensation, and the sphincters, and presents clinically the features of a cross-lesion of the cord of rapid onset. In extreme cases the patient falls dead, or, after staggering a little, falls and expires with or without delirium and convulsions. Symptoms may be most variously combined and show any grade of intensity. The majority recover promptly. Of those coming under treatment about one-half recover, about half have lasting paralysis, and about three per cent. die. Those who do not recover within the first three or four days present the symptoms and run the course of a lumbar or dorsal myelitis of varying extent and severity.

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[1] Hirt, "Handbuch der spec. Pathologie und Therapie," vol. i.

[2] "Berlin. klin. Wochens.," May 31, 1897.

[3] "Compressed-air Illness," London, 1896.

Treatment.—*Prophylaxis.*—Persons who are to be subjected to increased air-pressure should be rigidly examined. Those presenting the predisposing conditions mentioned should be excluded. Only hardy young men with sound hearts and regular habits should be accepted for this work, and spare individuals are preferable. In case of an extensive undertaking the same men should be employed throughout the task, that they may be habituated to the increasing pressure as the work progresses, thereby gaining immunity. In a pressure of over thirty pounds they should work short shifts of two hours or less, and plenty of time should be taken in passing the locks. Smith[1] says five minutes for each fifteen pounds of extra pressure. Inexperienced persons should take much more. It is well not to enter the pressure fasting. Snell lays stress on ventilating the works to reduce the amount of gases in the circulation as well as on general principles.

Treatment of the Attack.—If symptoms arise, the patient should be hurried back into the caisson, and if the symptoms subside, as they often do, very slowly returned to the outer air. Morphin is often required for the intense pain. Ergot in dram doses of the fluid extract every hour will sometimes relieve the pain and apparently check the disease. It has been suggested to bandage the limbs, and even the trunk, thereby restoring something of the surface pressure and maintaining the spinal circulation. When paralytic features have developed, the treatment is that of myelitis.

TUMORS OF THE SPINAL CORD AND ITS VARIOUS ENVELOPES.

New growths arising in the spinal meninges or on the inner surface of the bony canal or in the cord itself produce definite symptoms only as the cord or the nerve-roots are disturbed. They may be properly grouped together because of their common symptomatology, which renders a positive clinical diagnosis as to their original sites impossible, and makes every operation for their removal in a limited sense an exploratory one. The importance of an early diagnosis of these tumors is emphasized by the fact that most of them can be successfully removed if taken in time, and the fatality, which otherwise attends them, averted. The paraplegia to which they give rise may in the same way be prevented, and in some instances caused to recede when not too far advanced.

There are now in the literature less than 200 reported cases, so that tumors involving the cord are rare. Those arising from the meninges and extradural structures are about six times as numerous as those primarily cordal. Sarcoma and its varieties furnish more than one-third of the tumors, tubercle and echinococcus each about a tenth, and carcinoma and all varieties of benign growths, including gumma, the balance. Glioma, which is so common in the brain, also appears in the cord. It is usually, however, distributed closely about the central canal or in longitudinal bands in the substance of the cord, where, undergoing degeneration, it produces channels or false canals. These give rise to definite symptoms and a clinical type, which is called *syringomyelia.* It is described in a later chapter.

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[1] Pepper's "System of Medicine," vol. iii.

Tumors affecting the spinal cord are usually of small dimensions owing to the rapidity with which they destroy the cord and lead to a fatal issue. They are commonly single, but several tubercular growths and multiple sarcomata and neuromata have been reported. The favorite location of spinal growths is in the dorsal and lower cervical regions. Regarding the causation of spinal tumors, the same ideas exist as pertain to the development of tumors elsewhere. Traumatism is often alleged and may undoubtedly serve to locate a syphilitic process and perhaps to favor the invasion of tubercle and angiomatous growths. Its relation to malignant neoplasms is largely supposititious.

Morbid Anatomy.—The post-mortem examination reveals the new growths arising from the extradural tissues, the membranes, or the cord, in the substance of which it may rarely be embedded.

It frequently is traversed by several nerve-roots, or these may be destroyed. It may have a vertical extent of several inches. From pressure upon the cord there is a zone of softened cord-substance which frequently shows an inflammatory condition. The cord may be much indented by the growth, or compressed almost to complete division. Changes in the cord substance due to pressure-atrophy, softening, and myelitis are present with resulting secondary degenerations related to the portion of the cord that is affected. The consequences of myelic softening and myelitis are found in the muscles, bladder, kidneys, etc., depending in distribution upon the anatomical and clinical features of the given case. The various growths are marked by their ordinary individual characteristics and histological peculiarities.

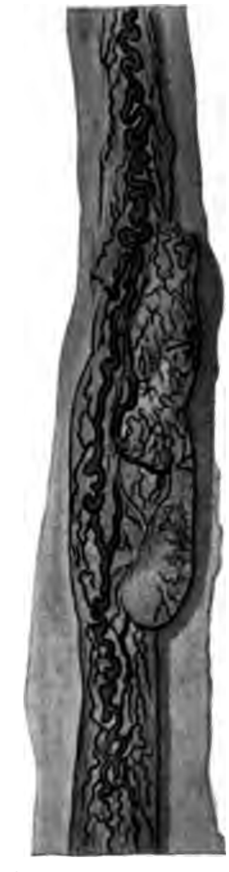
Fig. 136.—Tumor of the cord (Leyden).

Symptoms.—The initial and most common symptom of spinal tumor is usually *pain.* This is of two varieties: first, that referable to irritation of the posterior roots, and, second, that due to disturbance of the sensory tracts in the cord. The first gives rise to girdling sensations and partially follows in distribution the fields of the peripheral nerves. The second follows the segmental outlines and may also give rise to pains referred to parts below the lesion whose sensory pathways in the cord are irritated. If these pathways are broken, the pain may be referred to an area actually anesthetic. The root pains and the segmental or cord pains are frequently combined. Ordinarily the pain is bilateral. It may occur first on one side, but, arising as it usually does from pressure upon the cord, it is evident from the anatomical situation that pressure and counterpressure must be equal and the whole diameter of the cord soon affected. When the tumor is within the cord and laterally situated, a partial Brown-Séquard palsy may develop at first, but ordinarily soon yields to the cross variety and the paraplegic syndrome. The pains are of all degrees of severity, but frequently atrocious in their intensity, of a lightning, darting, ripping character, with paroxysms and remissions.

The *nerve-trunks* are so rarely sensitive that Starr,[1] in a masterly résumé of the subject, states that he never noted it in his own experience or in the literature. Pain over the seat of the tumor is also rare, and, when present, following the usual rule, is felt one or two inches below the level of the lesion.

The *reflexes* comport themselves as in the paraplegic state generally (see p. 363). Those which find their nuclear representation in the diseased segments are lost early. Those below the lesion are exaggerated as the compression is brought to bear, and, in extreme cases reach the highest degree of intensification. Should the cross-lesion of the cord become absolute, constituting a cord-division, all reflexes are lost below the affected level. The rectal and vesical reflexes are subject to the ordinary rules of the paraplegic state. *Motor disturbances* of a paraplegic sort rarely precede the sensory disturbance, but usually follow it *pari passu* with the reflex manifestations. The feet and legs feel heavy, clumsy, and weak. This paresis becomes more and more pronounced as the pressure increases; the spastic gait is developed, and finally the patient becomes bedridden. Absolute motor loss is the great exception and depends, like complete reflex obliteration below the lesion, upon division of the cord.

With the motor loss, again, there is usually blunting of cutaneous *sensation*, and finally complete anesthesia, of paraplegic distribution, surmounted by the hypersensitive band.

To again follow Starr, the order in which these symptoms arise is commonly: (1) Peculiar pains of limited distribution; (2) increase of reflexes below the lesion; (3) paraplegia; (4) loss of sensibility, and (5) loss of all subjacent reflexes. Bedsores and dystrophic joint-disturbance occur late in tumor, or may appear earlier upon the addition of an acute myelitis. Collins[2] names the clinical order of symptoms as follows: Sensory, motor, visceral, trophic, and topical; the topical consisting in tetaniform rigidities caused by pressure over the spine in the region of the tumor and in rare cases deformity of the spinal column.

Course and Prognosis.—The course of the disease is usually slow from the insidious onset to the fatal termination by exhaustion due to pain, or the consequences of myelitis, cystitis, pyelonephritis, bedsore, and septicemia. The rapidity of the disease depends upon the character of the growth, but tubercle is frequently of unexpected activity and may induce a complete paraplegia within a few weeks. In rare instances years have been consumed in the development of the tumor. The natural tendency is to paraplegia and death. The location of the tumor in the cervical or lumbar enlargement hastens the course of events.

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[1] "Am. Jour. Med. Sciences," June, 1895.

[2] "N. Y. Med. Rec.," Dec. 6, 1902.

The *prognosis* in tumor involving the cord, excepting gumma, is uniformly bad, and practically fatal without operation.

Diagnosis.—The diagnosis of cord-tumor depends mainly upon the insidious onset and the order of development of symptoms. The slow compression of the cord gradually induces the paraplegic state with intense spasticity, but is preceded by the localized, persistent, and uniform root or segment pains. A *cross-myelitis* is ordinarily of sudden onset and not especially painful unless the meninges are affected. In that event the pain is diffuse and intense over the spine. Bedsores and trophic disturbance are early features of myelitis, late ones of tumor. The presence or history of new growths elsewhere aids the diagnosis, and the location in the dorsal cord is of some significance. In *Pott's disease* we soon have rigidity of the back, pain upon rotation or percussion or jars of the spine, and later vertebral thickenings and deformities. The nerves are sensitive and the pain accurately follows the nerve-trunks as a rule.

The *location* of the tumor is deciphered by reference to the general rules of cord-localization, especial importance attaching to the location of the early pains and the upper levels of dysesthesia. As already indicated, it is usually impossible to clinically determine whether a growth is vertebral, meningeal, or cordal, and operations are, therefore, to this extent exploratory. A Brown-Séquard paralysis, followed by a paraplegia, or analgesia preceding anesthesia, or limited early muscular atrophy, may indicate that the growth is probably within the cord.

Speculation as to the *nature of the growth* is based on those general features which are common to growths in all localities. The presence of malignant growths elsewhere points to a secondary neoplasm of the same order. Tuberculosis and syphilis have their own indications. It may also be borne in mind that sarcoma is the most common form of tumor in this location.

Treatment.—With the exception of gummata, which are to a considerable degree amenable to antisyphilitic treatment, the only course that promises relief is surgical *operation*. It is evident that tumor within the cord-substance can not be removed without great and permanent damage to the cord. It is also evident that if the compression from without has caused local cord-disintegration, an operation can at best only check the harm at that particular stage. Malignant neoplasms are very likely to recur, and are frequently secondary to large growths elsewhere that have already jeopardized life. Tubercle is also often secondary to a process that may leave little hope of prolonged existence. The fact, however, remains that without removal the cord-tumor itself will induce a fatality, preceded usually by the most intense suffering and the most abject helplessness. Even when an operation can do nothing but relieve the pains due to the local irritation, it may be favorably considered.

On the other hand, about seventy per cent. of recorded cases were operable, and out of thirty-three operated cases, tabulated by Putnam and Warren,[1] over one-half resulted successfully. Similar figures are given by Starr and also by Bruns.[2] Early diagnosis and prompt operation will materially improve the average.

SPINA BIFIDA.

Spina bifida is an embryological defect due to the failure of the neural canal to completely close in the posterior median line. As a result, the bony arches of the vertebræ are defective. This may be indicated by a depression, *spina bifida occulta,* or by a tumor made up of the contents of the spinal canal variously arranged. Its usual location is in the lumbosacral region, as the neural canal closes from above downward and is last completed at the caudal extremity. When present, it serves to fix the lower end of the cord to the corresponding vertebræ, and the normal recession of the conus medullaris to the level of the second lumbar vertebra is prevented.

The simplest variety is the rare *meningocele,* which is usually covered by skin and consists of a pediculate or pedunculate sac of spinal membranes continuous with the arachnoid spaces. Neither the cord nor the nerves enter it, and deformities of the cord or disabilities in the legs and sphincters are absent.

The ordinary tumor in spina bifida is the *meningomyelocele,* made up of both the cord and the meninges and usually attended by deformities and paralysis in the legs and sphincters. The cord ordinarily is flattened out in the posterior surface of the tumor, which is more or less translucent, devoid of true skin, and only covered by a layer of epithelium. From this posterior situation the flattened and deformed cord-substance gives off the nerve-roots, which run forward through the sac into the intervertebral foramina. In the most prominent part of the tumor corresponding to the cutaneous defect and the location of the broadly spread cord-substance there is often a pit or umbilical depression.

In rare and more pronounced deformities the central canal of the cord dilates with the tumor formation, *hydromyelocele,* and lines its cavity, with which it is coextensive. The nerve-roots now lie in the sac-walls. Again, in *myelocele* a flattened mass—not a sac—of neural tissue lies in the vertebral hiatus, containing a small opening leading into the cerebrospinal canal. Through this, cerebrospinal fluid constantly oozes. Both of these forms are rare, always attended by defects in the lower extremities, and the latter, myelocele, is promptly fatal in a few days.

Etiology.—The causation of this defect in the embryo, which dates back to the earliest days of impregnation, like similar teratological defects, is obscure. It has been attributed to injury. In a number of instances it has appeared in several children in the same family, and even in succeeding generations, showing hereditary transmission. Other children in the same family may present harelip or club-foot. It is more common in females than in males, in a ratio of about eight to seven.

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[1] "Am. Jour. Med. Sciences," Oct., 1899.

[2] "Die Geschwülste des Nerven-Systems."

Symptoms.—Spina bifida is usually discovered at birth, but may exist unnoticed as a slight depression, often covered by hair for months or even up to adolescence, when the tumor may appear. At first usually small in size, it may rapidly increase. When the cord is involved in the sac, there is usually associated club-foot or undeveloped legs or sphincteric incontinence. The tumor is in the middle line and presents varying appearances due to its makeup. The opening in the vertebral arches can usually be detected by touch, but the contents of the tense, fluctuating, and slightly compressible sac are rarely palpable. Under excitement and in the erect position the sac increases in tension and size. Pressure upon it is likely to produce uneasiness, stupor, coma, and convulsions. The fluid contents correspond to and are identical with cerebrospinal fluid.

The diagnosis is usually very easy, but it is more difficult to decide upon the exact nature of the tumor's makeup. The presence of deformities or paralysis in the limbs or of sphincteric incapacity and umbilication would indicate that the tumor was not a simple meningocele, but that the cord was involved in the sac-wall. An oozing aperture and a nevus-like mass of pinkish tissue would suggest myelocele.

Prognosis.—Only in the slighter forms of spina bifida is the prognosis favorable. A pedunculated meningocele or mere depression is not incompatible with full vitality and perfect cord-functions. In the severer varieties, if the child lives, it will probably bear club-foot or other deformity and defect. The great majority of cases die during the first three months, cases of myelocele during the first three days. Rare cases have reached adult age and beyond.

Treatment is purely surgical. In meningocele a fair prospect is offered to extirpation of the sac. In the other and more common varieties, to extirpate the sac or to obliterate it by injections is to destroy a portion of the cord-substance. In this condition the sac must be preserved, and various osteoplastic operations have been suggested looking to the formation of a bony neural canal covered by cutaneous flaps. The deformities in the lower extremities and the paraplegic condition, of course, would not be benefited thereby.

CHAPTER III.

LESIONS PRINCIPALLY CONFINED TO THE GRAY MATTER OF THE CORD, AND DISEASES REFERABLE TO DISORDER OF THE SPINAL GRAY.

In this chapter a number of diseases are brought together that have in common symptoms dependent upon disorder of the lower motor neuron. In some, anatomical changes are clearly defined; in others there is reasonable presumption that eventually changes will be detected. The whole question of progressive muscular atrophy is in a transitional state, with a decided tendency to bring the various forms into close relationship.

ACUTE ANTERIOR POLIOMYELITIS.

Poliomyelitis anterior acuta, acute spinal paralysis, acute atrophic paralysis, spinal paralysis of children, and *essential paralysis of children* are the principal of the numerous names applied to an acute myelitis involving mainly the anterior horns of gray matter of the spinal cord and commonly appearing in children. It is marked by febrile and infectious symptoms, is of rapid onset and development, and promptly induces wide-spread muscular paralysis, a portion of which commonly remains permanently and is then attended by local atrophy.

Etiology.—The great majority of cases occur before the tenth year, and fully three-fifths under four years of *age*, with especial frequency in the latter half of the first year. During the first six months it is extremely rare or else passes undetected to a fatal end. It has been freely attributed to *dentition*, to *cold*, and to *traumatism*, especially in the form of falls, but it is doubtful whether any of these alleged causes are of direct importance. In many instances it occurs in the course of or during the convalescence from *infectious fevers*, especially the exanthemata. Its *infectious nature* is further indicated by the abrupt onset, the usual febrile movement, the gastric disturbance, the occasional occurrence of convulsions, and most of all by epidemic and endemic outbreaks. Such have been recorded by Colmer,[1] Cordier,[2] Medin,[3] Leegard,[4] Oxholm, Nonne, Calverly,[5] Altman,[6] and others. In some of these outbreaks a considerable variation from the type has been noticed. Some cases presented the symptom complex of Landry's paralysis, with the infectious nature of which we are already familiar; others evidence of acute polioencephalitis superior and inferior (see p. 153). If a neuropathic *heredity* counts for anything, it is in the vulnerability of the central nervous organs in certain individuals.

Morbid Anatomy.—The microscopical appearances, as well as the finer changes, vary greatly with the age of the lesion. The early descriptions based on recovered cases were misleading. At the end of *one or two months* a focus or several foci of myelitis are found in the anterior gray. There is local destruction of nerve-tissue, with dilatation and changes in the vessels. The ganglion-cells of the anterior horn involved in the lesion have lost their prolongations, show granular disintegrations, or have entirely disappeared. The myelitic focus occupies one or both of the two arterial areas of the anterior horn; namely, that area supplied by the first branch of the anterior median artery or that field supplied by the arteries which enter the cord along the anterior roots, or both. Rarely it extends backward in the middle arterial field and invades the neck of the posterior horn, and usually it embraces the adjoining white matter of the cord to a varying extent. It is evident that the lesion is not strictly systematized, and it is equally evident that it is vascular. A contention has arisen as to whether the lesion is first parenchymatous, as Charcot claimed, or is first interstitial, as insisted upon by Goldscheider, Redlich, and others. The present weight of authority points to invasion by the blood-vessels, interstitial dissemination, and cellular destruction as the natural order of events. Batten[1] asserts upon rather convincing evidence that the vascular fault is primarily thrombotic due to a variety of infections, and the lumbar cord is especially affected owing to the circulatory disadvantages of the part.

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[1] "Am. Jour. Med. Sciences," 1843.

[2] "Lyon Médicale," 1888.

[3] "Hygiene," 1890.

[4] "Neurolog. Centralblatt," 1890.

[5] "N. Y. Med. Record," 1891.

[6] "Australian Med. Gaz.," 1897.

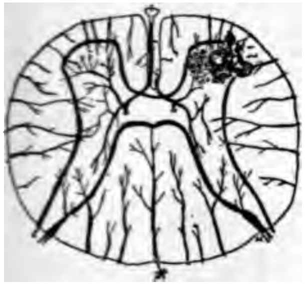

Fig. 137.—Diagram of arterial mechanism producing infantile paralysis (Williamson).

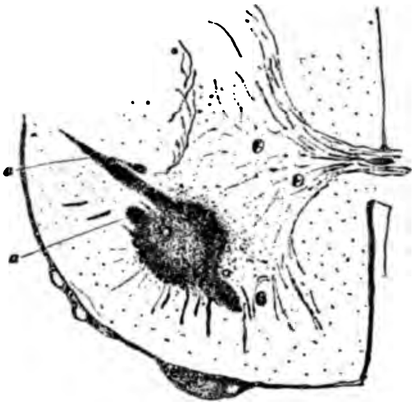

Fig. 138.—Section of spinal cord in a case of acute anterior poliomyelitis of the adult. *a*, *a*, Dilated vessels surrounded by round cells (Williamson).

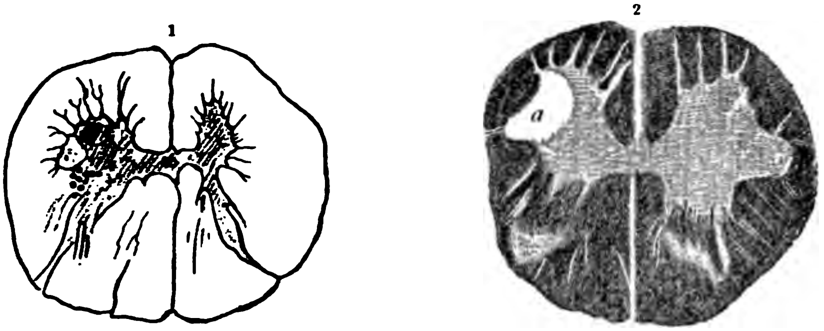

Fig. 139.—1, Section of cervical cord in an old case of infantile paralysis. The right half is shrunken in all its extent (Charcot). 2, Lumbar section in a case of infantile palsy: *a*, Focus of old inflammation; left half of cord shrunken.

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[1] "Brain," Autumn, 1904.

The myelitic foci vary in extent and number in various cases. Sometimes they extend through several segments of the cord, eight or ten centimeters or more in a vertical direction, but usually are one to two centimeters in longitudinal extent. Sometimes single, more frequently there are several foci either on the same or on opposite sides of the cord and at various levels. They may even invade the medulla, and the brain is not absolutely exempt.

In cases of *many years'* standing the cord is seen to be shrunken on one side at the level of the lesion and the cross-section is lacking in symmetry. The acute inflammatory condition has long disappeared and a fibroid condition alone represents it. Nerve-fibers and cells are entirely destroyed. The absence of myelinated fibers renders the scarred area more or less glossy and translucent to the naked eye. The vessels are thickened and appear more numerous on that account. The shrinking of the lateral half of the cross-section involves mainly the gray matter and especially that of the anterior horn, but the white columns and the posterior cornu may also share in it to some extent. If the original lesion is sufficiently extensive to involve the spinal apparatus of an entire extremity, or even of a segment of it, changes in the cord above the lesion, and in the brain, are found analogous to those induced by amputations.

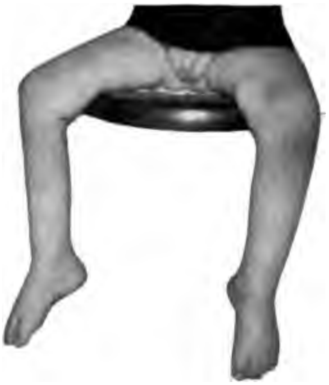


Fig. 140.—Case of poliomyelitis affecting the lower extremities unequally; foot-drop on both sides. Note the wasting of the right leg and thigh.

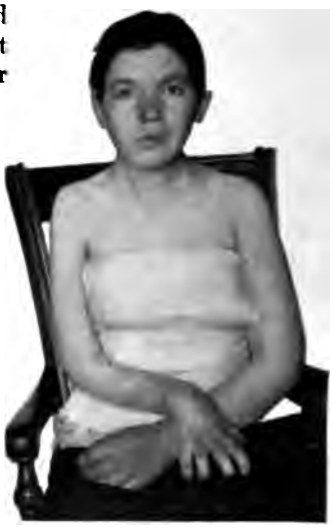


Fig. 141.—Case of poliomyelitis affecting right face (rare), right arm, and shoulder.

The *anterior roots* arising from the injured part of the cord are often diminished in volume and may or may not show alteration of structure. This is true equally of the mixed *nerve-trunks*. The exact condition depends apparently upon the age of the lesion and the complete or partial disappearance of the peripheral prolongations of the spinal pyramidal cells that have been injured. In the paralyzed *muscles* we have the same changes that are found after division of the nerve-trunk, but a few muscular fibers often persist, some of which may even show hypertrophy. The rest of the muscle is fibrous and fatty. Infiltration of fat sometimes reaches a condition that may properly be designated lipomatous. The *bones* in the paralytic members are retarded in their growth, and in old cases dating from childhood are undersized in comparison with those of the sound side. They lack the sharply defined muscular markings, and the Haversian systems are found to be undersized and poorly developed.

Symptoms.—Usually without apparent definite provocation the child is found to be feverish and ill. A *temperature* of 100° to 102° F. has been frequently noted, and this febrile invasion stage lasts from a few hours to several days, when *paralysis* and *flaccidity* of one or more limbs are detected. It is not rare, however, for the child to go to bed apparently well and to awake paralyzed in the morning. The febrile movement may be attended by vomiting and diarrhea, by *convulsions* of a generalized character, or by *delirium* and diffuse cerebral manifestations. As soon as the paralysis is noted, the case is usually recognized. Most writers state that there is a complaint of pain in the afflicted members only rarely, but that, as a rule, *sensation* in all its phases is entirely normal. It is probable, however, that early dysesthesia, owing to the usual infantile age of the patient and a lack of careful search for such difficulty, has been frequently overlooked. In some considerable number of cases handling of the affected limbs during the initial fever provoked outcries which were not elicited by similar manipulation of the other members. It is likely that more attention in this direction will show localized hypersensitiveness or some kindred state to be usually present and of diagnostic importance. Indeed, complaints of pain and of formication have been generally noted in older children and in adults, lending, perhaps, undue weight to the usual supposition that such cases are not of a true spinal type. The sphincters are seldom relaxed, so that control of the bladder and bowel remains unimpaired, but in the rare cases in which the sphincters are relaxed there is more or less apparent loss of sensation, the extent of the lesion is greater, and the prognosis is extremely unfavorable.

Even in fat children the implicated *muscles* can be seen, after a few weeks, to have wasted, and if tested with the faradic current, either do not respond at all or show a remarkable diminution in their excitability. At this time the patient will have begun to show considerable improvement, the motor paralysis remaining complete only in the parts that are to suffer permanently, and a gradual improvement may be expected to extend over several months. In the muscles showing lessened faradic excitability galvanism commonly produces exaggerated responses, as compared with the sound limbs, and the complete reaction of degeneration or any modification of it may be encountered. In a well-marked case faradism fails by the tenth day, and the increased galvanic response appears, lasting for about six months, when it gradually fails. At this point faradic excitability returns and the muscle regains something of its size and strength; or, if too seriously impaired, faradic response does not reappear, galvanic response disappears, and the muscle is irretrievably lost.

The *reflexes* are lessened or abolished in proportion as the muscles which are anatomically associated with them are involved, or perhaps it would be better to say that their alteration depends upon the implication of the cornual cells making up a part of their arc. *Bones* which have not attained their full growth are retarded or entirely fail to develop if their trophic centers are implicated.

The seriously atrophied muscles become fibrous bands, and, since they offer neither assistance nor opposition to the synergic and antergic muscles, *distortions* soon develop, with *joint-changes* and sometimes *subluxations*. Joints which depend upon muscular support, as the shoulder, may allow of so much deformity by the relaxation of the muscles which have lost their tonicity that the articular surfaces widely separate. The *skin* is inactive, often cold, and sometimes dry and scaly, but the atrophic conditions so usual in neuritis are practically absent and bedsores are almost unknown.

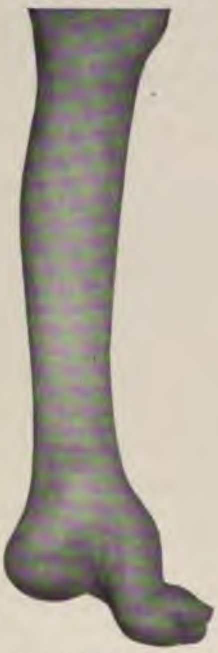


Fig. 142.—Deformity of foot after lumbar poliomyelitis acuta.

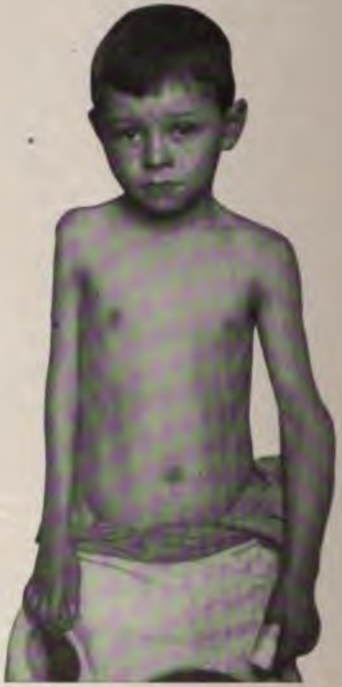


Fig. 143.—Deformity of upper extremity and thorax after poliomyelitis anterior acuta.

The lower extremities are affected about three times as frequently as the upper, and the left leg twice as often as the right. A crossed form, in which the upper extremity on one side is involved with the opposite lower limb, is not rare; but involvement of both limbs on the same side is extremely uncommon. In the *lower extremity* the extensors seem more susceptible than the flexors; hence drop-foot, with talipes equinus, flexed knee, and flexed thigh are common. When the paralysis is below the knee, the sural muscles usually escape. In the *upper extremity* the most frequently encountered wasting is in the small muscles of the hands, the deltoid, and the extensors of the wrists. The biceps and supinators generally escape.

The distribution of the paralysis of muscles follows their segmental relations in the cord, and, consequently, when limited is of a functional character. The very fact of such functional distribution at once incriminates the cord. The muscles of the trunk are in rare cases notably and widely affected, producing weakness in the back, deviation of the spinal column, and defects in the muscles of the chest. Ordinarily, however, the segments of the cervical and lumbar enlargements are selected. A small focus of anterior poliomyelitis in the dorsal region hardly produces local signs, as the trunk and thoracic muscles are represented in a considerable vertical extent of the cord. In rare cases, however, the abdominal or chest muscles or those along the spine are involved very decidedly. In a less degree this is not rare, as shown by spinal twists, protuberant abdomen, and abnormal respiratory movements of the thorax.

The bulbar muscles do not always escape, as is shown by various ophthalmoplegias, squints, facial and hypoglossal palsies, which are now and then recorded, and would be more often seen did not coincident pneumogastric accidents usually terminate such cases fatally.

Course and Forms.—The course of the disease may be clinically divided into: (1) A stage of febrile invasion, lasting from a few hours to several days, with local tenderness and rapidly developing and increasing paralysis; (2) a stationary stage, lasting for several weeks; (3) a period of improvement, lasting to the end of a year, and (4) a stage of permanent disability for the remainder of life. Relapses during the early weeks have been recorded in very rare instances, and second attacks are still rarer. Among the sequelæ the contractures, dislocations, and deformities have been already mentioned. The fragility of the bones makes them liable to fracture, but union takes place with ordinary promptness under proper fixation.

The *adult form* is in no way different from the infantile cases, excepting the variations due to complete growth having been attained. In anomalous cases the onset of the disease is insidious and the course may be subacute. After infectious diseases one or more limbs may be found useless, flaccid, and atrophic, declaring the antecedent myelitis. Occasionally the onset is marked by pains of a severe, cutting character, which are frequently attributed to neuritis or to rheumatism, but may be due to the irritation of the sensory pathway in the cord by the location of the myelitic focus backward, involving the neck of the posterior horn.

It has been noted by a number of observers that in rare instances, years after the acute disease has subsided, a slow, insidious *reappearance* has led to a progressive muscular atrophy, which declares itself first in the limbs that were originally paralyzed by the acute process. This may progressively invade the entire muscular system.

Diagnosis.—In the early stages of fever, before paralysis has appeared, diagnosis usually fails except under rare epidemic conditions. The termination of an apparently trivial ailment in extensive paralysis is frequently the cause of much chagrin on the part of the medical attendant, who may naturally enough have expressed a favorable prognosis. As already indicated, the initial fever may be readily mistaken for that of general disorders, and sometimes, though rarely, the pain in the limbs leads to the idea of rheumatism. If, however, the possibility of anterior poliomyelitis be in mind, and examination discloses some slight local tenderness or diminished muscular activity, or both, a guarded opinion will naturally follow. It is only when the paralysis is developed or developing that the nature of the disease becomes certain, and even then, if there have been cerebral symptoms, such as delirium or convulsions, difficulties are not at an end. The *cerebral palsy* of children is almost invariably ushered in by convulsions, but these have a definite distribution involving one side or one limb, or only the face, while the convulsions of the disease under consideration are generalized. Localized pain, from *traumatism* or inflammation, may cause immobility of a limb, and when preceded by fever gives rise to a doubt; but the usual absence of extreme sensory disturbances in disease of the anterior horns is a distinguishing feature, while the local conditions can be otherwise made out. After a few days the electrical test gives absolute data. Faradic response is abolished in no other disease so early; even in severe *neuritis* it is longer maintained, and is then attended by a very marked sensory disturbance, which also usually precedes it for a long time. A few careful applications of the induced current at this period can do no harm. The progressive infantile *myopathies* are of extremely slow development and are symmetrical in distribution.

Prognosis.—So far as life is concerned, this disease terminates fatally only exceptionally, and if the patient survives the onset of the paralysis but a short time, life may be considered out of danger. Further, one can say, with a reasonable degree of certainty, that the paralysis at first developed will notably recede, but, unfortunately, it is almost equally certain that a portion of it will permanently remain. At the end of a fortnight a carefully conducted faradic examination of the muscles enables the physician to speak more specifically regarding the amount of permanent disability. At that time any muscle which responds, however feebly, may be expected to regain a fair degree of its former tone and strength. Even after several months, repeated faradic stimulation of a muscle, at first perfectly inactive, may develop some contractility, and this is of favorable import for the given muscle. The extent of permanent paralysis governs the amount of resulting contracture and deformity; and, likewise, the retardation of development of the limb and of the bones is in similar relation. The anticipated amount of these deforming conditions will have a bearing on the probable general activity of the individual, his prospective physical health, and liability to fractures. The possibility of the late appearance of a progressive muscular wasting may be kept in mind.

Treatment.—Owing to the irregularities of the course of this disease in various cases, and its natural tendency to improve up to a certain point, it becomes a matter of great difficulty to estimate the value of any therapeutic agent or mode of treatment. In the early stage, as soon as the diagnosis is made, if fever still continues, there is good reason to suppose that antipyretic antiseptics, like the salicylates, large doses of the tincture of the chlorid of iron, or even bichlorid of mercury, would do good. Hot applications can be made to the spine if the circumstances of the patient will insure their intelligent and faithful employment; otherwise they are worse than useless, and very mild sinapisms can be more properly used. The child should be kept on the side or face, and the affected limbs should be thoroughly enveloped in cotton-wool to maintain the circulation and the nourishment of the muscles in the parts laboring under diminished trophic influence. The use of spinal stimulants like strychnin while the lesion is active is to be avoided; but when the active process has come to a standstill,—that is, ordinarily, at the end of a fortnight,—its systematic use is one of the most important measures. The usefulness of electricity consists of maintaining the nourishment and normal contractility of the muscles, which are temporarily deprived of their natural motor and trophic control, so that, as the inflammation subsides and the wide-spread inhibitory effect of the local lesion recedes, the central apparatus may find the muscular periphery in the most favorable state to respond to its enfeebled influence. For this purpose, as the faradic response is early abolished, the interrupted galvanic current must be used, the slightest intensity being employed that will cause a contraction. Care must be taken not to unduly fatigue the muscles. A dozen contractions at most should be elicited at one séance, and often only one or two can be provoked by any strength of current that is bearable. Care not to alarm the child is imperative, as a daily struggle will do more harm, probably, than the electricity will do good. As the muscles often react better and with less pain to the positive pole than to the negative, it is well to have for the negative electrode a broad sponge which may be placed on the sacrum or breast, and with a smaller, positive sponge the muscles may be exercised. Applications of galvanism through the cord are quite useless, and even if such currents reached the lesion, which is doubtful, their effect for good is questionable.

Later on, as faradic response returns in the muscles only slightly affected or temporarily inhibited, this form of electricity is efficacious for the purpose of local stimulation, and the presence of this reaction in any muscle is always, as already indicated, a gratifying circumstance. To intrust galvanic electrical treatment to the parents, however intelligent they may be, is a mistake.

In the same way local frictions and salt baths, warm wrappings, and massage are valuable measures which can be more rationally trusted to parents or nurses who take an intelligent interest in the work. They may be gently employed from the first day, and when electricity is not tolerated or can not be systematically employed, must be relied upon to replace it. The moment a group of muscles weakens, the limb tends to assume an abnormal position, and it is very important to meet this tendency from the very first moment, even in cases where there is every probability that the paresis will recede. It can be easily accomplished by means of the warm wrappings, or even by the application of light splints. There can be no question that recovering muscles will find their task much easier if their proper relations have been maintained. Unbalanced muscles will be much less liable to contractures if an artificial balance has been provided and joint-surfaces have not been altered by long-maintained vicious positions.

As soon as the permanent paralysis can be fairly well foretold, massage should be especially directed to obviate the contractures and deformities that ordinarily result. Stretching of the unopposed muscles by passive movements of the joints will accomplish much, and the moment a tendency to contracture is perceived, the case becomes one for mechanical appliances. The tendency to talipes equinus, for instance, can be met by a light elastic cord from the toe of the shoe to a band at the knee; and more elaborate orthopedic apparatus should be employed at the knee and hip if required. These cases are, therefore, practically orthopedic disorders from the very first.

Nearly all the improvement that is to take place in the muscles will have developed by the end of the first year, and what is slowly subsequently gained, seems independent of any treatment whatever.

The treatment of a late or neglected case is practically surgical. Shortened tendons may be cut and joints straightened. Tendons of healthy muscles may sometimes be transplanted to take the place of those paralyzed. Much is being accomplished by nerve suture. The involved nerves are being grafted into sound ones and the innervation of the atrophic area re-established. A resection at the knee is sometimes of advantage to secure a rigid limb instead of a useless contortion or a dangle-leg. By using a high shoe or other appliance crutches may often be laid aside. The arrangement of exercises to increase the strength of the involved muscles which retain some fraction of their muscular elements is of distinct advantage. By intelligent passive and active movements and effort against resistance much can be accomplished.

SYRINGOMYELIA.

The central canal of the cord is sometimes enlarged through congenital defect. This may be a part of a general ventricular distention, causing hydrocephalus and spina bifida. In certain instances unattended by any symptoms during life a tubular cord has been found post mortem. These cases present *hydromyelia* and are of teratological origin. The term *syringomyelia* is now limited to acquired enlargement of the central canal or to the formation of entirely new canals of considerable length in the gray substance of the cord. Such canals are the result of gliomatous infiltration about the central canal or in the gray horns of the cord and its subsequent degeneration, forming tubular, cystic cavities. In rare instances a central myelitis or a central hemorrhage may cause a fusiform or tubular excavation that can not always be clinically distinguished from the neoplastic variety or may in turn induce the latter. Some cases probably originate in spinal hemorrhage occurring at birth. About 300 cases have been reported and many more have been observed.

Etiology.—Men are much more frequently affected by this disease than women, and especially men exposed to hard labor. Verhagen and Vandervelde report several instances of syringomyelia in the same family, but a neuropathic heredity is rare. Adult years furnish nearly all the reported cases, but it has been observed well developed at nineteen by Langdon, of Cincinnati, by the author at sixteen, and by Ballard and Thomas at three.[1] Cold, rheumatism, exposure to bad weather, traumatism, overwork, the acute infectious fevers, and syphilis,[2] have been reported as possibly causative in various cases, but this relation, in the gliomatous form, at least, is entirely conjectural.

Morbid Anatomy.—The syringomyelic cord in marked cases shows notable changes of conformation that correspond to its tubular condition. It is soft, flattened, and sometimes fluctuating. The collapse of the canal may produce a furrow, so that the cord seems double. The condition finds its favorite locality in the cervical region, but may extend throughout the entire length of the cord or be confined to any portion of it. Its upward extension carries it into the fourth ventricle.

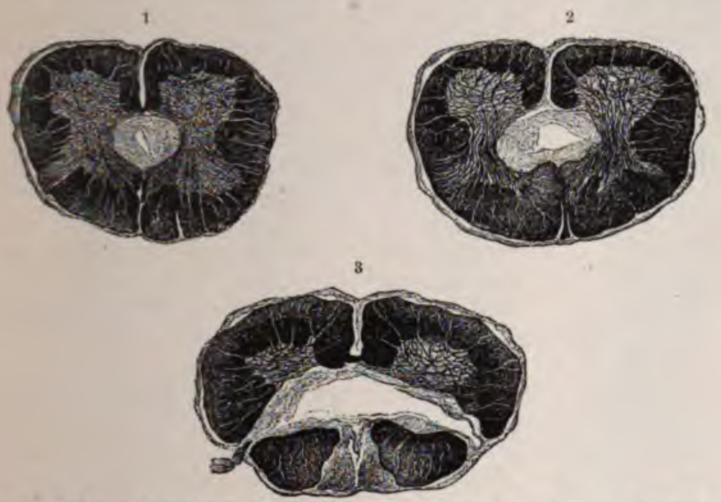
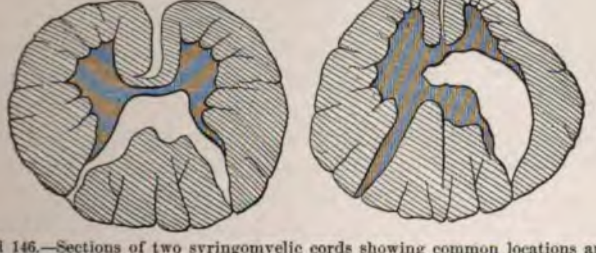


Fig. 144.—Sections of a syringomyelic cord. 1, Lower lumbar region; 2, upper lumbar region; 3, midcervical region (Bruhl).



Figs. 145 and 146.—Sections of two syringomyelic cords showing common locations and extent of cavities (Brissaud).

The cross-section usually reveals a single oval cavity, or there may be several in communication or independent. The customary situation is in the immediate neighborhood of the central canal and behind it, but it may extend laterally into the anterior or more commonly into the posterior horn of gray matter, either on both sides or unilaterally. Again, it may be limited to one posterior horn. While the white columns of the cord may be spared, it is not unusual for the gliomatous process to invade them, especially the pyramidal tract, when the gray matter has been widely destroyed and the cavity has attained large proportions.

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[1] "Am. Jour. Med. Sci.," Mar., 1899.

[2] Nebelthau, "Zeit. f. Nervenh.," Feb., 1900.

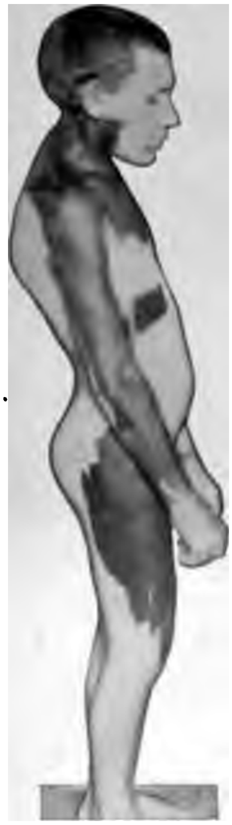

Fig. 147.—Case of syringomyelia with areas of thermo-anesthesia marked in black. There is cervical kyphosis.

The cavity contains cerebrospinal fluid, which is sometimes bloody or gelatinous. Its walls are made up of a well-defined substance, outside of which the cord appears infiltrated, dense, and even sclerosed. In the medulla, when the lesion extends so high, the gray matter is first similarly affected.

The microscope shows the usual new formation to be gliomatous and rich in blood-vessels, especially on the external periphery. The predominance of various elements gives rise to varieties such as pure glioma, neuroglioma, and vascular glioma, all of which may be present in the same cord. Schultze describes an infiltrating sort as a gliosis. It does not usually result in cavity formation, and is, therefore, distinguished from the gliomatous variety. He is supported by Hoffman, and Holt and Harter[1] report a case. Orlowski[2] has seen a double canal, one arising from dilatation of the central canal and the other from gliosis and degeneration outside the central canal. Charcot, Hollopeau, Joffroy, and Achard insist that in some cases the initial step is a central myelitis, which Schultze, Dejerine, and others deny. The nerve-tissues involved in these various processes are strangled and destroyed even before the formation of cavities. The cells first yield their prolongations and then disappear. The axis-cylinders outlast the myelin. The meninges and spinal roots are usually unaffected except in the form associated with cervical pachymeningitis. Destruction of the anterior horns is followed by the usual trophic disturbance in the associated periphery, such as wasted muscles, perforating ulcers, and cutaneous dystrophy. The interference with the cells of Clarke's columns is supposed to account for the joint-lesions that are commonly present, and which are identical with the arthropathies of locomotor ataxia.

Symptoms.—From the nature of the lesion it is apparent that the symptoms may in different cases embrace perversion and obliteration of all the various functions of the cord. Nevertheless, their peculiarities and groupings are sufficiently distinct to frequently enable a diagnosis. As there is no common type, the symptoms must be arranged somewhat arbitrarily.

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[1] "Am Jour. Med Sci.," April, 1895.

[2] "Arch. de Neurol.," Sept., 1898.

Sensory Disturbances.—The early involvement of the sensory pathways in the gray commissure and in the posterior horns and columns produces related sensory symptoms which are well-nigh characteristic. There are usually definite coextensive areas of thermo-anesthesia and analgesia, with retention of the tactile sensibility. This is sometimes denominated the *syringomyelic dissociation* of cutaneous sensation, and is highly important to the diagnosis. The patient fails to distinguish the temperature of water or objects brought in contact with the skin, though recognizing contact, and may receive burns in this way without experiencing any pain. The thermo-anesthesia may be complete or partial, and is then relative to the part examined and somewhat to the patient's intelligence (see Part I). Sometimes heat is felt as pain, or "hot" as "cold." The distribution of the thermo-anesthesia is also significant. Rarely it may be hemiplegic; it is seldom general. Ordinarily, it involves the limbs, or portions of them, such as would be covered by a glove or sleeve, a sock or long stocking. It may be limited to thoracic or abdominal bands. It may be unilateral or symmetrical. In figure 147 it is of unusual outline, but will be noticed to roughly conform to some spinal segments. In some cases areas at first discrete have been observed to coalesce into anatomical cord-territories.

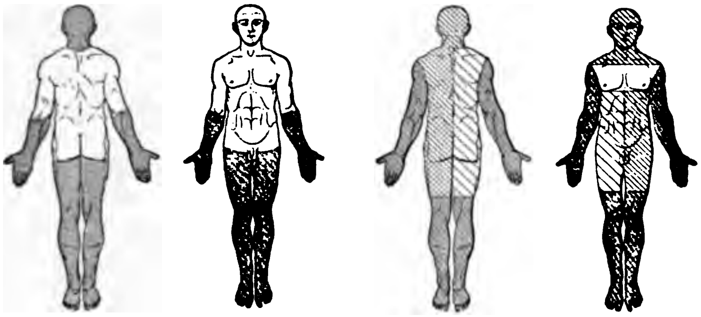

Fig. 148.—Various distributions of thermo-anesthesia and analgesia (Brissaud).

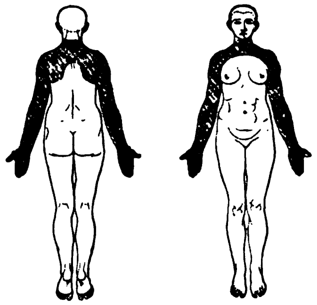

Fig. 149.—Thermo-anesthesia and analgesia (Brissaud).

The *analgesia* may be partial or complete, and usually conforms in outline to the thermo-anesthetic area or may be more extensive. It embraces all the tissues, so that felons, caries of bone, and disintegrating joints may be painless and insensitive.

As a general rule, *tactile sensibility* is perfect. A slight diminution of it is not inconsistent with the diagnosis, and its obliteration follows extreme invasion of the posterior columns, being then a part of the absolute anesthesia that marks such a lesion. An anesthetic area may thus have a border of thermo-anesthesia and analgesia, or these conditions may be found in different parts of the body, in relation to the amount and distribution of the cord disease.

Motor disturbances are dependent largely upon the invasion of the anterior horns and the pyramidal tracts. They are, therefore, usually secondary in point of time to the sensory symptoms with which they come to be associated. As the anterior horn is invaded, the associated muscles lose power and their reflexes diminish and disappear. Some *muscular atrophy* is almost always present, and it may reach a high grade. Its distribution depends upon the part of the cord involved, and may conform to many of the typical spinal atrophies, such as the form of Duchenne-Aran, for which it has no doubt been frequently mistaken. It may be progressive or advance by spurts, and is most common in the upper extremities. Fibrillary twitching and reaction of degeneration mark the atrophic muscles, as in other muscular atrophies of spinal origin, and their strength is reduced in proportion to the atrophy. When the gliomatous process invades the lateral tract, spastic features are induced. These may be associated with disorders due to involvement of the anterior horn, producing the conditions found in amyotrophic lateral sclerosis, but the face is usually unaffected. Involvement of the posterior columns causes ataxia and incoördination.

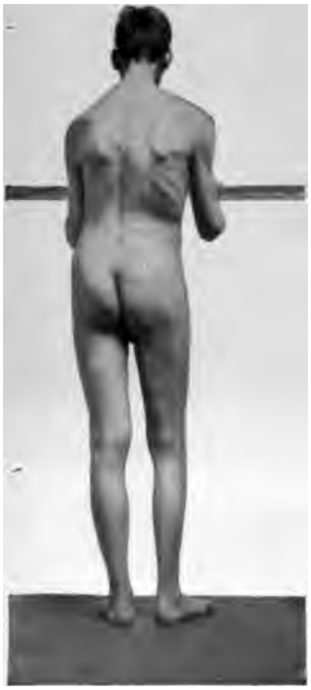

Fig. 150.—Case of syringomyelia, showing atrophy over right scapula and thorax and in right leg. Spine scoliotic.

Trophic Features.—The affected extremities, in addition to the muscular atrophy, often present trophic disturbances, especially about the digits, similar to those in neuritis. Glossy skin, hypertrophic nails, increased or diminished perspiration, and herpetic and bullous eruptions are encountered. Cuts, burns, and abrasions heal badly or tend to permanent ulceration. In this way paronychia causes the nails to fall. Felon is rather common, especially in the Morvan type, and causes mutilations of the fingers by the loss of several phalanges, unattended, usually, by the slightest pain. Perforating ulcer is encountered with some frequency. Boils, abscesses, and other local infections are not rare. They all heal badly, produce extensive scars, and may cause mutilations and deformities.

The *arthropathies* are almost invariably represented. They affect the spine by preference, and the articulations of the upper extremities more frequently than those of the lower limbs. In some cases the bones are affected. They are fragile, readily fractured, and unite with difficulty and with persisting callous deformities. In a few cases the hands have been enlarged, as in acromegalia, for which this disease has been mistaken. They have even been found associated. The spinal arthropathies give rise to deviations of the vertebral column in over one-half of the cases. Usually it is a scoliosis, but angular deformities are not infrequent. The muscular weakness may account for some of the spinal deformities. A deep cavity at the upper portion of the chest in front has been noted several times by Astie [1] and Schrader has reported two cases presenting habitual dislocation of the shoulder due to changed contours of the humeral head and the glenoid cavity.

Vasomotor symptoms are represented by dermographia, blueness or redness, edemata, and localized disturbance of perspiration, especially in the affected areas.

Unusual Symptoms.—In various rare instances the following unusual and rather accidental conditions have been present, due to the special location of the gliomatous disease in the particular case: Loss of sphincter control, sexual impotence, suppression of menstruation, pupillary inequality, narrowing of the palpebral fissure and retraction of the eyeball, nystagmus, facial paralysis, hypoglossal paralysis, optic neuritis, pneumogastric accidents, and bulbar invasion, producing a progressive bulbar palsy and other cranial-nerve disablements.

Course.—Syringomyelia is a chronic malady of slow progression and fatal termination. It often presents stationary periods or even slight temporary improvement may be noticed. Bulbar invasion means early termination. It has exceeded forty years' duration in favorable cases, and may be interrupted by death from concurrent or accidental disease. Its logical termination is in death by exhaustion or by bulbar crises. Ulcerations, dystrophic conditions, or infections from such sources may induce a fatal termination.

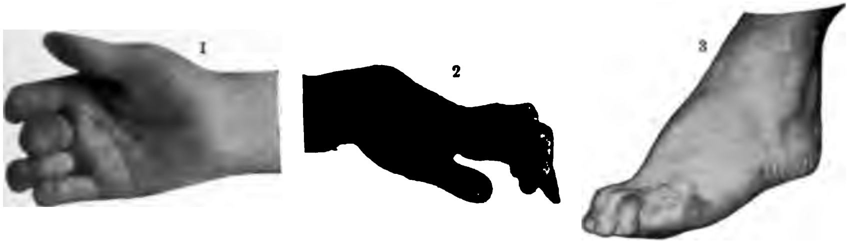

Fig. 151.—Hands and foot in cases of syringomyelia of Morvan's type, showing, 1, mutilations of the fingers from whitlows, 2, osteo-arthropathy of wrist-joint, and 3, loss of toes.

Clinical Forms.—The ordinary form has been in view in the preceding lines. One clinical variety is furnished by *Morvan's disease.* In this form the sensory dissociation is marked, especially in the hands and arms, with associated atrophy and paresis. There are mutilations of the fingers by successive whitlows and marked cutaneous dystrophy. Scoliosis and arthropathies are usually present. Zambaco insists that this is a form of leprosy, and Prus [2] has demonstrated in such a case a bacillus closely resembling that of leprosy. On the other hand, Joffroy and Achard have demonstrated central cord-cavities in Morvan's disease, and this is confirmed by Thomas.[3] In both a peripheral neuritis is common, and Prus suggests that the propagation of the leprous bacilli in the cord might furnish the irritation that incites the gliomatosis. The question remains open.

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[1] "Thèse de Paris," 1897.

[2] "Archiv f. Psychiatrie," Bd. xxvii.

[3] "Rev. Méd. de la Suisse Romande."

There is more than an accidental relation between syringomyelia and a cervical *hypertrophic pachymeningitis*. Brissaud[1] insists that the meningeal process usually precedes and furnishes the irritation that sets up the central gliomatosis. It seems probable that any permanent cord irritation may lead to a secondary syringomyelia.

Prognosis.—The outlook in any given case is ultimately fatal, but the duration of the disease must be estimated by the course it is running and the portions of the cord affected. Bulbar symptoms are extremely grave and lumbar involvement is hardly less so. The tendency to stationary periods and slight remission must, however, be kept in mind.

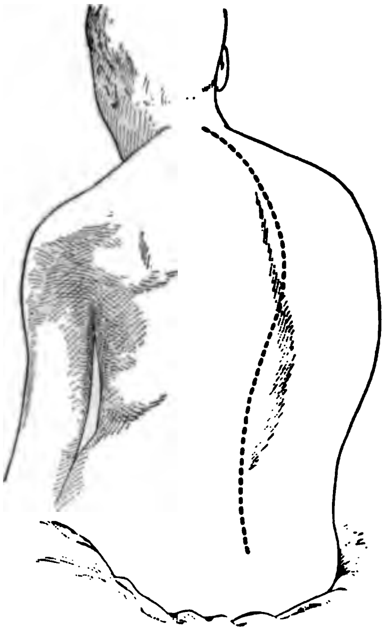

Fig. 152.—Scoliosis in Morvan's type of syringomyelia (from a photograph). The spinous processes were marked with iodin.

Diagnosis.—The diagnosis depends upon the insidious development of the disease and upon the combination of sensory, trophic, and motor disorders. The dissociation of touch and pain is well-nigh distinctive, but is occasionally found in tabes, neuritis, and hysteria. Usually it will be necessary to exclude progressive muscular atrophy, amyotrophic lateral sclerosis, pachymeningitis hypertrophica cervicalis, locomotor ataxia, and peripheral neuritis. Perhaps acromegalia, scleroderma, leprosy, and hysteria may at times confuse, but in all these an attentive study of the combinations of symptoms should enable a diagnosis to be made.

Treatment has proved futile, or nearly so. Measures looking to the general well-being of the patient are most advisable. Locally, massage and electricity may give slight help at times. Counterirritation over the spine must be used with caution, as it is capable of producing extensive and rebellious ulceration. Surgical measures are likewise contraindicated because of the dystrophic element in the affected parts.

PROGRESSIVE MUSCULAR ATROPHIES PRESENTING LESIONS OF THE SPINAL GRAY MATTER.

All progressive muscular atrophies may be divided into those in which (1) lesions of the spinal gray matter are found, and (2) those in which no such changes are discoverable by our present means of investigation. It may at once be admitted that this division is arbitrary and probably temporary. It is not unlikely that many members of the second group will eventually be found in the first. The present conception of the integral character of the lower motor neuron, embracing the spinal pyramidal cell, its peripheral prolongation, and the muscular organ under its motor and trophic domain, is inconsistent with the idea that the so-called muscular dystrophies or idiopathic muscular atrophies are independent of disturbance in the spinal gray. For purposes of convenience they will be separately described, though every gradation is found from first to last, and even a single case may present several of the varieties at one or in successive periods.

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[1] "Leçons sur les Maladies Nerveuses," 1895.

Duchenne, followed closely by Aran, in 1849 and 1850 described a progressive muscular wasting without sensory disturbance, which they called progressive muscular atrophy and thought it a disease of the muscles. Cruveilhier, in 1855, recognized it as a spinal disease, and Lockhart Clarke first limited the lesion to the anterior spinal gray matter. Charcot still further differentiated the lesion and pointed out its dependence upon degeneration of the ganglion-cells of the anterior horn. Later on Charcot distinguished a variety of progressive muscular atrophy in which the lateral tracts were also degenerated, and called it *amyotrophic lateral sclerosis.* The first variety is now called by French writers *Duchenne-Aran's disease,* the second, *Charcot's disease.* Of late, some writers, notably Marie, have tended to deny the existence of the Duchenne-Aran type in toto. Others, as Gowers, insist that both the Duchenne-Aran type and the type of Charcot are identical, only varying as the lateral tracts or the anterior gray—that is, as the upper or lower motor neurons—are first or most degenerated. Gowers states that he has never seen a case in which lesions in both spinal regions could not be detected, and cases presenting lesions confined absolutely to the anterior gray no longer appear in current literature. Many cases formerly classed with the Duchenne-Aran type were doubtless those of multiple neuritis, syringomyelia, Charcot's disease, and the so-called idiopathic muscular atrophies to be later described. We may, therefore, properly discard many of these conflicting terms and speak of progressive muscular atrophy with or without cord-lesions.

Attention is now directed to the first sort, which embraces conditions variously called *spinal progressive muscular atrophy, wasting palsy, chronic poliomyelitis, amyotrophic lateral sclerosis, atrophia muscularis progressiva spinalis, Duchenne's disease,* and *Charcot's disease.*

Etiology.—Progressive spinal muscular atrophy is practically a disease of adult life occurring between the *ages* of twenty-five and fifty, but it may appear earlier or later. It is somewhat more frequent in *men* than in *women.* Direct *inheritance* is very rare, but a neuropathic tendency can occasionally be traced. It has been noted as following various alleged vague causes, such as anxiety, overwork, exposure to cold, concussion of the spine, syphilis, and various infectious and septicemic states. Its appearance years after an *acute poliomyelitis* has been already indicated under that caption. A close inquiry will sometimes detect a history of symptoms antedating the alleged cause. There can be little doubt that at least in some instances it is an expression of teratological defect in the motor and trophic portions of the central apparatus, and constitutes a primordial shortcoming by which these parts reach an early death.

Morbid Anatomy.—The lesions of progressive spinal muscular atrophy embrace in rare cases the entire motor field of the nervous apparatus from cerebral cortex to muscular nerve-endings, and include the muscles themselves. Both upper and lower motor neurons in their entirety are destroyed by a degenerative process. Following the pathological rule that a neuron degenerating from toxic cause or involution first shows changes in its peripheral portion, the upper motor segment may present alteration only in the pyramidal fibers of the cord. This may reach the medulla, and, as a rule, does not extend into the peduncles, capsule, and cortex, though it may do so. In the lower neuron the degeneration is probably at first peripheral, but in all cases that reach a marked development the cells of the anterior gray are found degenerated. Attending this we have muscular atrophy, with fibroid and fatty changes and degeneration in the motor fibers of the nerve-trunks, limited sharply by the anatomical relations of the diseased cord-elements.

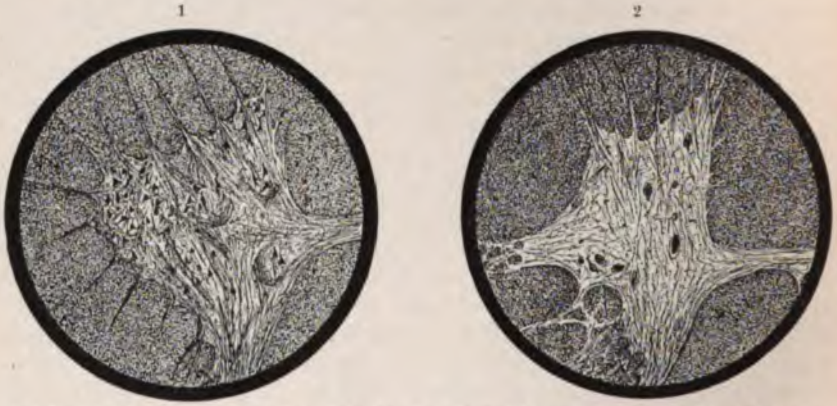


Fig. 153.—1, Normal anterior horn; 2, anterior horn in a case of amyotrophic lateral sclerosis (Marie).

In the *cord* the *gray substance* of the anterior horns shows atrophy. The ganglion-cells, many of which usually have disappeared, are wasted and degenerate, and there is a general shrinking of all the nervous elements of the horn. The *white substance* of both the direct and crossed pyramidal tracts shows sclerotic degeneration. This process is not strictly confined to them, but usually involves the anterolateral tracts to a lesser degree, and may invade the lateral limiting layer. This is especially the case in the upper dorsal and cervical regions. The columns of Goll sometimes show slight changes, apparently due to the shrinking of the myelin, and not to an active degeneration. The lesions of the anterior cornua are generally most pronounced above the dorsal region, but the involvement of the crossed pyramidal tracts extends to the lowest cord-levels. Similar changes may be traced through the *medulla*, both in the gray matter below the fourth ventricle and in the pyramidal tracts above the decussations. These latter may mount through the *peduncles* and *internal capsules* to the pyramidal-cell layers of the *cortex*, and there a cellular degeneration, identical with that in the spinal gray, may occasionally be encountered.

The *muscles* are pale and fatty, and under the microscope present various changes. The fibers may be narrowed, with striæ poorly marked or less frequent than in health, or more frequent and deeply marked, showing a tendency to fissuration. The striation may entirely disappear in fatty granulations, and distinct globules or the empty sarcolemma sheaths may alone remain. Longitudinal striation sometimes develops, and the transverse markings may later disappear. The *nerve-trunks* often contain degenerated fibers, which can be traced through the anterior spinal roots to the anterior horns. All these central and peripheral changes vary in degree in different cases, and intermediate instances are becoming more and more frequently noted in which the muscles or the nerve-endings or the anterior cornual cells show preponderating changes. Only the most approved technic can be relied upon to determine minor abnormalities.

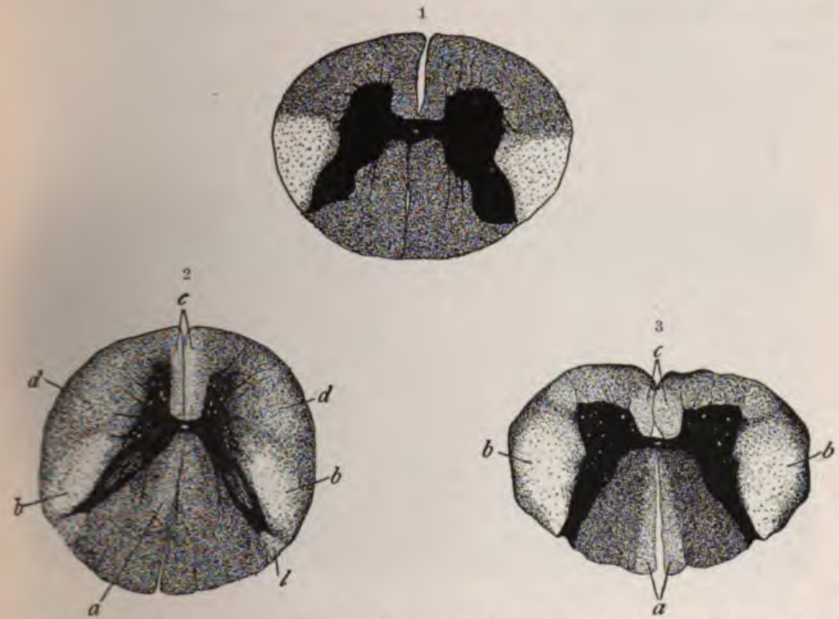


Fig. 154.—Cord-sections in a case of amyotrophic lateral sclerosis. 1, Lumbar region; 2, dorsal region; 3, cervical region (Marie).

Symptoms.—The various types of spinal muscular atrophies and the confusion that has arisen over them, as well as the symptoms in various cases, are referable to the vertical extent of the lesions and the varying intensity of the process at different levels. It must at once be evident that if the degeneration falls first on the lower neuron, flaccid, atonic, atrophic paralysis will appear in the periphery. On the other hand, if the upper neuron be primarily affected, the paralytic state will be spastic and the tonic atrophy will be marked by rigidities. All degrees between these extremes are encountered in practice. Flaccidity may be present in the upper and spasticity in the lower extremities in the same case. Involvement of the upper neuron can only be evidenced through the lower in the way of increased reflexes and spasticity. If, then, the lower neuron leads the upper in the degenerative course, the latter can not produce its signs. Again, the degenerative process falls by preference on the cervical cord and manifests itself in the upper extremities first. Thence it tends to extend, and eventually reaches the cranial nuclei in the bulb, inducing a progressive bulbar palsy. In other cases the higher levels are first affected, presenting bulbar palsy or ophthalmoplegias, and the downward extension of the disease may be cut short at any point by pneumogastric accidents. Labiolinguopharyngeal paralysis or bulbar palsy is apparently identically the same disease first affecting the bulbar centers. It is also variously combined with the spinal forms.

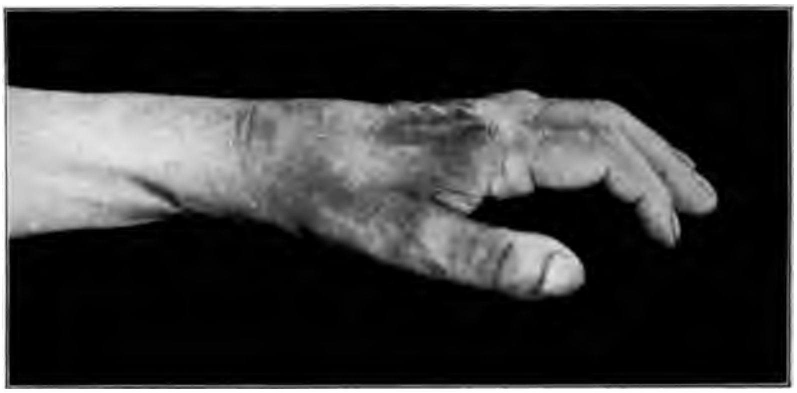


Fig. 155.—Hand in advanced case of amyotrophic lateral sclerosis, showing muscular wasting and the characteristic ape position of thumb.

The muscular atrophy and weakness usually come on together and progress equally. In the great majority of cases wasting appears first in the radial half of the hands, sometimes first in one hand, sometimes in both hands at once. In another group of cases the shoulder-muscles are first affected. In the hands the disease ordinarily affects first the thenar muscles. It, therefore, appears to elect those muscles which show the highest differentiation of function and which represent the latest motor acquirements in the evolutionary scale,—namely, circumduction of the upper limb and the opposition of the thumb to the fingers. If the morbid process be one of involution, we might naturally expect such a program. It results in flattening the palm, and the thumb falls back into the same plane with the other digits, producing the "ape hand" (Fig. 155). The interossei and lumbricales are also affected, and again most on the radial side of the hand. Furrows between the metacarpals mark the loss of the small muscles, and the integument hangs loose and redundant with numerous folds and wrinkles over the bones, entirely denuded of muscular covering in severe cases. A persistence of subcutaneous fat sometimes obscures the muscular atrophy. In late cases there is a marked tendency to clawing of the fingers, from the involvement of the small muscles, so that the first phalanges are extended, the second and third sharply flexed (Fig. 155). They may be rigidly and spastically fixed, all voluntary motion destroyed, and passive motion much limited. The muscles of the forearm suffer next, or they may escape for a time and the shoulder-muscles waste first. In other cases the muscles of the neck are first involved, and then those of the upper extremity, or the wasting may even develop first in the legs. Muscles like the pectoralis, deltoid, and trapezius, which have different associations, and receive their innervation from different spinal levels, may waste in corresponding portions. It thus happens that the upper portion of the trapezius is frequently spared until late in the disease, as it is controlled by the spinal accessory. Again, it may suffer early. The muscles of the back are among those early invaded, and those of the scapula are commonly first affected, allowing corresponding displacements of this bone and limit-

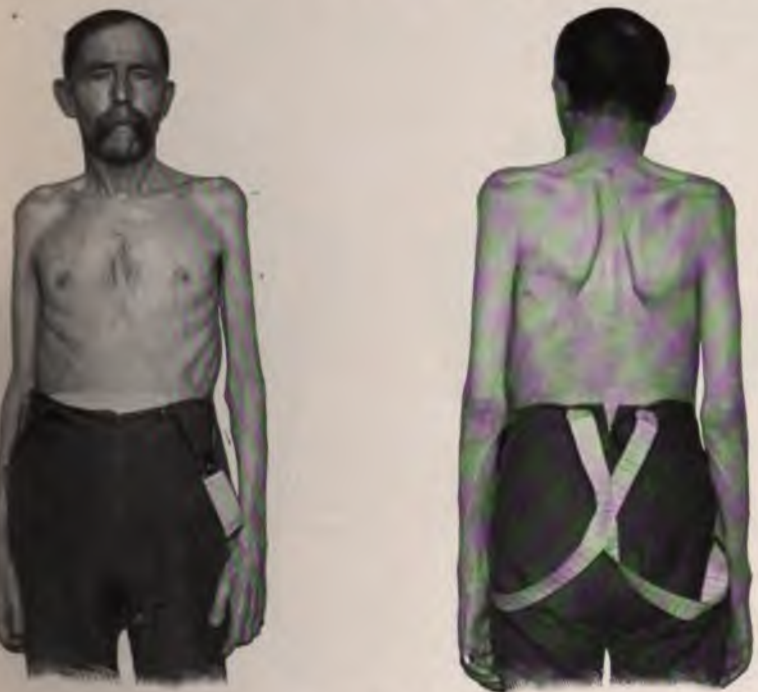
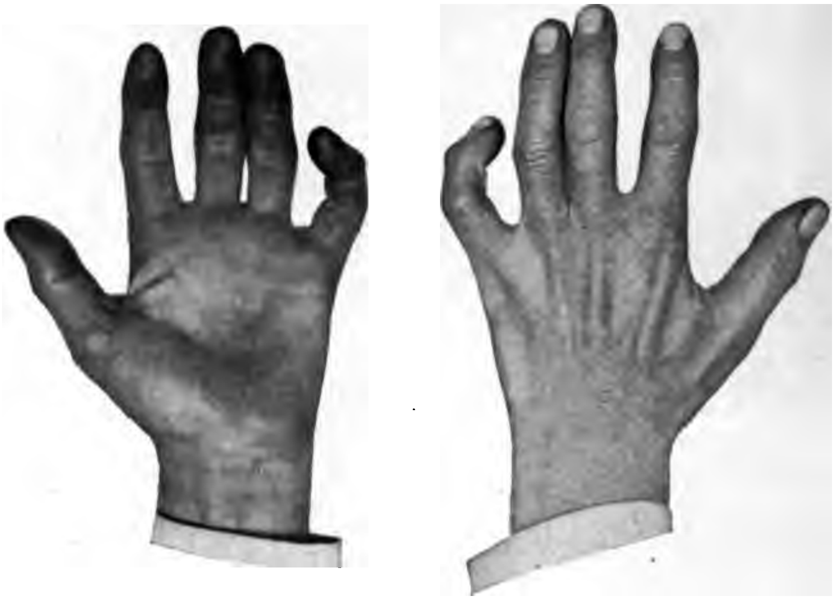


Fig. 156.—A case of progressive muscular atrophy of the spinal type in the sixth year of the disease. All skeletal muscles affected.

ing the use of the arm. The thoracic muscles are also involved, impairing respiration, and if the abdominal groups suffer, breathing may become purely diaphragmatic. Wasting in the legs is much less common and is usually less in degree, though atrophy may here first show itself. In the same way the face escapes for a time, or the disease may commence in the bulbar nuclei, and labioglossopharyngeal palsy may be the first step toward generalized progressive muscular atrophy. Finally, no voluntary muscle may escape.

The muscular wasting and loss of power result in corresponding changes of contour and position deformities. The patient may be unable to hold the head erect. The uncovered acromium renders the shoulder angular. The claw-hand, the scoliotic spine, the displaced scapulæ, and the distorted chest manifest the unsupported skeleton. The characteristic of the atrophy is its gradual invasion of a muscle fiber by fiber, commonly preceded by *fibrillary twitchings*. These twitchings are sometimes decidedly exaggerated, causing arhythmic jerking movements of the hands and arms or twitchings of facial muscles.



Figs. 157 and 158.—Hand in early amyotrophic lateral sclerosis, showing excavation of the interosseous spaces and flattening of palmar eminences.

The *reflexes* may be either abolished or more commonly increased in activity. Early and rapid wasting is often marked by diminution and even extinction of the reflexes in the affected muscle; but, again, there is often early rigidity and marked myotatic irritability, which last throughout the disease and persist even when the muscles are much wasted, or reflexes at first exaggerated may disappear. The reason for this variability has been already indicated.

In the lower extremities, however, it is the rule that the reflexes are augmented and spastic disturbance of a paraplegic sort is commonly found unless the atrophy begins in the lower members. This gives rise to the spastic gait often marked by clonus and dragging footsteps. The rigidity may show itself by the fixity of position, even when the patient is seated or in bed. The legs tend to extension, the arms to demiflexion, and the hands are held in the negative position, midway between pronation and supination. The *sphincters* are seldom affected. An excessive *jaw-jerk* can sometimes be elicited, and implies bulbar extension of the disease. The *cutaneous reflexes* are normal so long as their muscles survive the atrophic invasion.

The *bulbar symptoms* of the disease correspond to those of bulbar palsy (see p. 153). Fibrillary twitchings about the mouth and eyes and in the tongue are followed by wasting, and the characteristic facial expression is developed. In advanced cases the open mouth, the druling saliva, the difficulty of deglutition, the nasal voice, and the pneumogastric palsies only too plainly indicate the upward extension of the disease and the critical condition of the patient. The pharyngeal reflex usually persists as long as the pharyngeal muscles have power to act. The mind is not disturbed.

The *electrical response* in nerves and muscles is, as a rule, quantitatively reduced for both currents, and finally extinguished. In a rough way it is proportionate to the amount of muscular fiber present. In most instances the reaction of degeneration is found in a few muscles, or any variation of it may be presented.

Sensibility is practically intact. In some instances there is complaint of dull pains at or before the onset of atrophy, and vague feelings of weakness, fullness, and formication may be mentioned during the disease. The paralytic and wasted limbs are usually cold and the circulation is poor, but trophic disturbance in the skin, perforating ulcers, arthropathies, and bedsores are unknown. The visceral functions are not notably impaired.

Varieties.—The chief varieties to be distinguished are: First, cases marked by flaccid, atonic atrophy, which reaches an extensive degree, usually first appearing in the small muscles of the hands,—the Duchenne-Aran type; second, cases marked by similar wasting, but less in degree, and presenting tonicity, rigidity, and increased reflexes from the first,—amyotrophic lateral sclerosis, or Charcot's disease; third, an intermediate variety with only slight muscular wasting, but with great weakness and with spasms and retained reflexes; fourth, the variety beginning as a labioglossopharyngeal paralysis, an ophthalmoplegia, or some bulbar palsy.

Course.—These spinal muscular atrophies may commence in various ways and first invade any portion of the cord or the bulb. The clinical course varies accordingly. It is, however, progressive, and may terminate in a few months or a year, or consume twenty or thirty years in its evolution. Apparently stationary periods in the protracted cases are not uncommon, but the logical termination in all is death from cardiac or respiratory failure. Intercurrent disease, especially acute infections and particularly pneumonia, are badly borne and frequently end life.

Diagnosis.—In fully developed cases of the spinal progressive atrophies the diagnosis is usually not difficult, but a differentiation from the so-called *idiopathic muscular atrophies* of the next section may sometimes be impossible. We rely upon the presence of fibrillary twitching, increased reflexes, if only in the legs, and the reaction of degeneration in at least some of the muscles, to indicate the spinal location of gross changes. These cases usually begin in adult life and show no particular family history; the second group usually begin in childhood and are frequently familial. The spinal varieties select the muscles of the hands, shoulders, or lips first; the idiopathic varieties commonly spare the hands at first and may select the humeral muscles or the peroneal group first, and are frequently marked by pseudohypertrophies, especially of the calf, gluteal, and scapular muscles. *Multiple neuritis* ordinarily affects all four extremities and can be traced to some competent infection or poisoning. In it the sensory symptoms are prominent from the first, while they are slight or absent in spinal myopathies. *Syringomyelia* may produce a local atrophy, but has its distinctive index in the dissociation of cutaneous sensations and the usual presence of arthropathies and other trophic disorders, besides usually being unsymmetrical. *Multiple arthritis* is sometimes marked by extreme muscular atrophy. Here we have the early history of articular disease and the continued existence of arthritic mischief. The wasting, too, is usually on the proximal side of the joints, thus sparing the small muscles of the fingers. It is as likely to appear in the lower as the upper extremities, is rarely accurately bilateral, and affects first and principally the joint extensors. The reflexes may be increased, if not inhibited by pain or ankylosis, and the faradic irritability is usually increased. *Transverse myelitis* is limited distinctly by an upper border of sensory and anatomically corresponding motor disturbance. The onset and clinical history are acute.

Prognosis.—The prognosis is always grave, and in proportion as the disease tends to invade the bulb death is imminent. Intercurrent affections present more than their proper danger.

Treatment is practically futile, but should none the less be conscientiously instituted. The continuous use of nitrate of strychnia, as recommended by Gowers, in increasing dose, for long periods of time by the hypodermic method should be faithfully tried. Beginning with $\frac{1}{30}$ grain, three times a day, the dose may be gradually increased, under proper supervision, to $\frac{1}{10}$ grain at a dose and even to $\frac{1}{5}$ grain in some cases. The application of the thermocautery to the back, the careful use of exercises, massage, and electricity to the muscles, and endless attention to the general health and the processes of nutrition and elimination may prolong life. The danger of choking to death or of inducing an aspiration pneumonia when the pharynx is involved must be borne in mind. It is to be hoped that some product, perhaps of biochemistry, may be discovered that will maintain vitality in the degenerating motor apparatus and postpone the involutional changes of the disease.

PROGRESSIVE MUSCULAR ATROPHIES NOT MARKED BY STRIKING HISTOLOGICAL CORD-LESIONS.

The view-point regarding a group of clinical forms that have in common the feature of progressive muscular weakness and atrophy, often associated with hypertrophy or pseudohypertrophy of some muscles, has altered markedly during the past few years. When it was first found that such cases presented no spinal lesion, they were termed *progressive muscular dystrophies* by Erb, and *primitive progressive myopathies* by Charcot. A variety of forms were described by different observers, whose names, unfortunately, became associated with and served to fix these variations, which are now, following Erb, Brissaud, Sachs, and others, grouped under a common head. Writers also refer to the *pseudohypertrophic* form, the *brachial, facioscapulohumeral, pelvic,* and *peroneal* types, as hypertrophy prevails or the atrophy is most pronounced in the various indicated regions. As material has accumulated, transition forms have been encountered with increasing frequency. Two or more of the foregoing types have been found in the same patient or in members of the same family. What is more important, the same family has presented cases of progressive muscular atrophy of the spinal form and also of the so-called idiopathic muscular variety in one or in succeeding generations. Again, there is an increasing number of observations going to show that in the so-called myopathic cases the spinal gray is not absolutely normal. A much larger series of observations discover changes in the muscle nerve-endings or in the peripheral extremities of the lower neurons. The so-called peroneal or neurotic type furnishes an intermediate form between the spinal and the alleged purely muscular varieties. This is clinically indicated by the fibrillar twitchings, the reaction of degeneration, the relation of the muscular dystrophy to certain spinal segments, and the functional relation of the affected muscles. Yet similar segmental and functional outlines are presented by all cases. This alone compels an acknowledgment of the spinal factor. An attentive and intelligent study of any given case will usually show the features of several of the so-called types and serve to prove their essential identity. The upper spinal levels or the musculature pertaining to the upper spinal segments are first invaded in some; in others, the lower portion of the cord first manifests the disease.

If any significance is attached to the autonomy of the lower motor and trophic neuron, it will be impossible to consider the cell-body in the spinal horn as above reproach when its axonal prolongation shows degenerative changes, or when its trophic control is perverted or destroyed. At present such change in the cell may be called dynamic or functional when no morphological alteration can be discovered, but the pertinent fact remains that actual changes appear in some instances, and the probability is strong that improved technic will discover them in all. Sainton[1] has found well-marked changes in the spinal cord in the neurotic form, and Abadie and Denoyes,[2] in a typical pseudohypertrophic case, obtained the electrical reactions of degeneration that are supposed to be a part of spinal and nerve disintegration. Indeed, as before insisted, every gradation, from progressive spinal muscular atrophy to cases only showing muscular changes, can be adduced. Some observers, however, insist that the spinal changes are secondary to the muscular atrophy,[1] and well-examined cases still fail, in some instances, to present any demonstrable cord variation, even when the clinical type suggests the spinal variety of disease.

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[1] "Nouv. Icon. de la Salpêt., June, 1899.

[2] "Nouv. Icon. de la Salpêt.," Aug., 1900.

These *muscular atrophies*, to adopt that term for convenience only, have in common a familial tendency. They often appear in several members of a given family or in blood-relations of the same or different generations. While they may appear at any age, they show a marked tendency to occur in the early years of life, and commonly affect the roots of the extremities rather than their distal ends. They are almost always unmarked by changes of sensibility, by fibrillar twitchings, and by the electrical reactions of degeneration.

Etiology.—Regarding the causation of these progressive myopathic atrophies very little can be positively stated. Their appearance in several members of the same family or in several members of succeeding generations, their interchangeability of form, their propagation by the females, their appearance during the age of active growth and at developmental epochs of life, stamp them as hereditary, familial, and *embryonic*. Meek has demonstrated that new-born animals possess many more muscle fibers than adults, as, for instance, in the biceps, and Pick [2] suggests that in some instances this natural disappearance of muscle fibers may, from prenatal or other cause, exceed physiological limits and produce progressive dystrophy. The flabby and wasted muscles of old age may be in part due to a similar cause. *Age.*—The great majority of cases appear before puberty is established, with especial frequency during the second dentition and at pubescence. Others are first manifest during adolescence, from eighteen to twenty-six years of age; and a rapidly diminishing series is encountered later in life. It sometimes happens that, appearing at adult years in one generation, the myopathy follows in early childhood in the next. As males are rendered impotent by the disease, its transmission necessarily falls to those females of the family who escape and reach maturity. In addition, *boys* seem more frequently affected than *girls*. Appearing commonly during the years of childhood, it often follows the diseases peculiar to that period of life, but it is difficult to assert their causative activity. In some cases diphtheria has seemed to lead to it; in others it has followed exposures to cold, slight injuries, and infections. Given a neuron feebly endowed with enduring qualities, it is not improbable that any condition capable of reducing the general health, or any toxic state may act upon it with unusual virulence.

Morbid Anatomy.—The *muscles* show various morphological changes, depending upon the presence of hypertrophy, pseudohypertrophy, or atrophy,—the final and logical end for all the affected muscles. The sarcode elements may be hypertrophied or atrophied. Enlarged fibers may be found in a mass of greatly wasted muscular tissue. There is round-cell infiltration, vacuolation, splitting, division, and longitudinal striation of muscle-fibers, with hyperplasia of connective tissue and an increase in fat that may reach the highest degree of lipomatosis. The early changes consist of increase of connective tissue, possibly some muscular hypertrophy, then muscle atrophy and fatty infiltration. What appears clinically as a large muscular mass, in some instances is found to be a markedly lipomatous structure almost devoid of muscular fibers.

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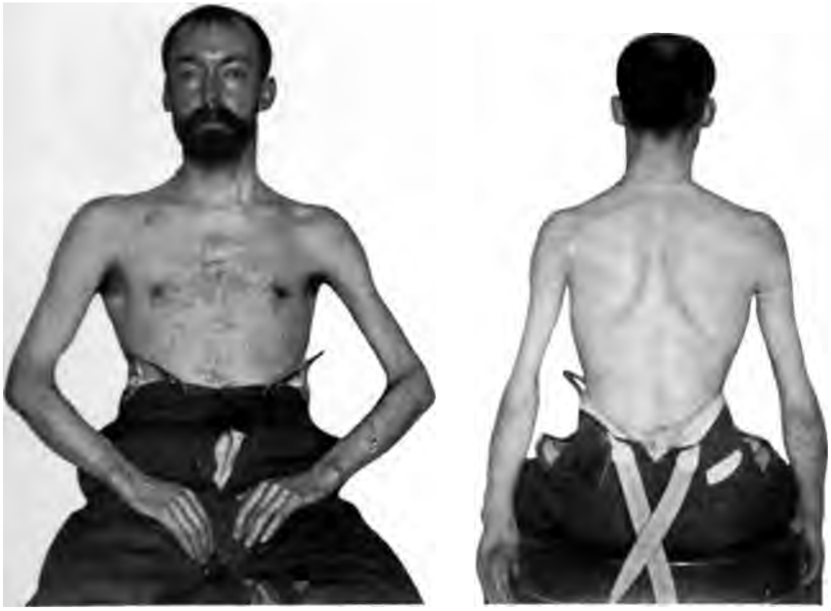
[1] Pick, "Deutsch. Zeit. f. Nervenh.," Bd. xvii, 1900.

[2] Pick, *loc. cit.*

Von Babes[1] has found the muscle *nerve-plates* undeveloped and degenerated. Heubner, Strümpell, and Marinesco have described the *peripheral nerves* as degenerated in the peroneal or leg type of the disease; the muscles and the anterior pyramidal cells of the cord were also involved. Hoppe has found similar changes in the facioscapulohumeral variety. Sachs and Brooks[2] have observed universal shrinkage of the posterior root ganglion cells in one case. To ordinary reagents the nerves and pyramidal cells usually react in a normal manner, but the finer technic of recent years is likely to determine changes in both.



Figs. 159 and 160.—Scapulohumeral type of progressive muscular atrophy. Note the angle on right side of neck with lengthening of the neck, due to wasting of the trapezius, and the peculiarity of the deltoid.

Symptoms.—The most striking symptoms depend upon the *changes of contour* in muscles and their loss of strength. These are variously combined in different cases. We will first take them up in systematic order, and finally group them in describing the common types of the disease. The *myopathic facies* depends upon the paresis of the facial muscles. The face presents a vacuous, sleepy, inert expression, and fails to adequately show forth the lively emotions that may actuate the patient. The brow is smooth, and the frontalis is unable to assist in raising the thin and drooping eyelids. The wasting of the eyelid muscles may give the eyeball a false appearance of prominence and cause a haggard expression. The lower lid, from the laxity of its tissues, often tends to droop. The cheeks and lips are flaccid, and the lower portion of the face droops from the bones. The eyes can not be firmly closed nor the lips puckered. In some cases the lips are thickened by hypertrophy and drag down, increasing the nasolabial furrows. The mouth may then remain open constantly, with a thickened, pendent lower lip. In other cases the lips are thinned, motionless, and can not be retracted from the teeth. In either case puffing, whistling, or spitting is poorly performed or impossible. The movements of the eyeballs are usually not disturbed, but in two cases the author has noted a very great loss of conjugate lateral movements. Movements of the tongue may also be greatly limited. The palatal excursions may likewise be reduced in amplitude, and laryngeal disturbance may be added.

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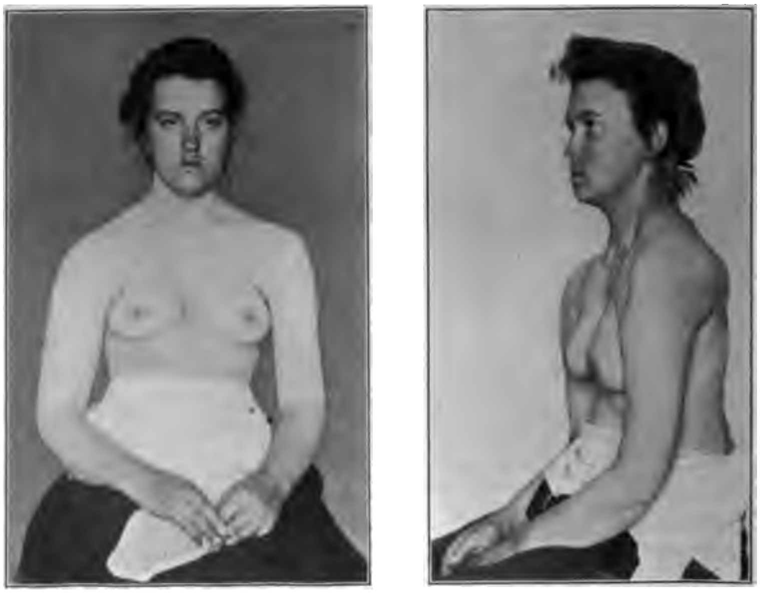
[1] "La Semaine Méd.," Aug., 1894. [2] "Amer. Jour. Med. Sciences," July, 1901.



Figs. 161 and 162.—Case of facioscapulohumeral type, showing facies, tapir mouth, horizontal clavicles, etc.

Brissaud calls particular attention to an apparent lengthening of the *neck* that he finds a constant symptom, and due to the drooping of the shoulder. The direction of the *clavicle* is frequently outward and downward, instead of upward, as in health. This may be accentuated by the early involvement of the trapezius, causing the characteristic angle, as shown in figures 159 and 160. The drooping shoulder may also appear only on one side. Attempts to extend the arms laterally, or even to hold them rigidly at the sides, cause a widening of the neck by the prominence of the upper borders of the trapezii. The *supraclavicular hollows* are thereby inordinately exaggerated and the sternomastoids spring into great prominence. Even the upper inner angle of the scapula may become salient above the normally curved line from mastoid to acromion. The *scapula* are usually mobile, and may wing out freely from the chest. The *spinati* muscles, especially the infraspinati, have a marked tendency to pseudohypertrophy, which may add greatly to the scapular deformity and accentuate the anterior curve of the lumbar *spine* when viewed from the side. This curvature is very commonly present, and may reach extreme proportions. It is due to the weakening of the spinal erectors in part, but is increased greatly when the glutei allow the *pelvis* to tip forward on the heads of the femurs, and is dragged into still greater prominence by the protuberant *belly*, due to the weakened abdominal muscles.

The *deltoid* requires particular mention. It is commonly hypertrophied and shows the marked discrepancy between the bellies and extremities often found in long muscles. The fibrous changes are marked at the origin of the muscle from the scapula and clavicle, apparently displacing the full, round shoulder-cap to the outside of the neck of the humerus, where it stands out as a lumpy mass (see Figs. 159 to 177). Similar changes in contour are occasionally encountered in the biceps of the arm, in the anterior crural, and in the sural group. The forearms and hands are commonly spared, at least until very late in the disease, but K. Mendel [1] has reported a case showing early involvement of the interosseal muscles of the hands. The *glutei* and the *gastrocnemii* are favorite locations for pseudohypertrophy. The calf-muscles also are frequently shortened so that the patient can not raise the toes from the floor while standing on his heels nor can the foot be passively flexed beyond a right angle. This interferes with the gait, causing the patients to stumble over the smallest obstacles. It may even produce an equinovarus. In rare cases, as in the one illustrated on page 406, a general increase in the adipose tissue may greatly obscure the

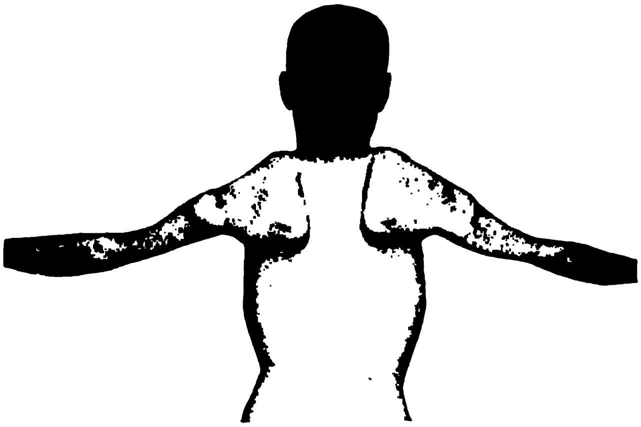


Fig. 163.—Progressive muscular atrophy at advanced stage, showing deformities about shoulders, with pseudohypertrophy of infra-spinati, wasting of all dorsal muscles, and spinal anterior curvature.

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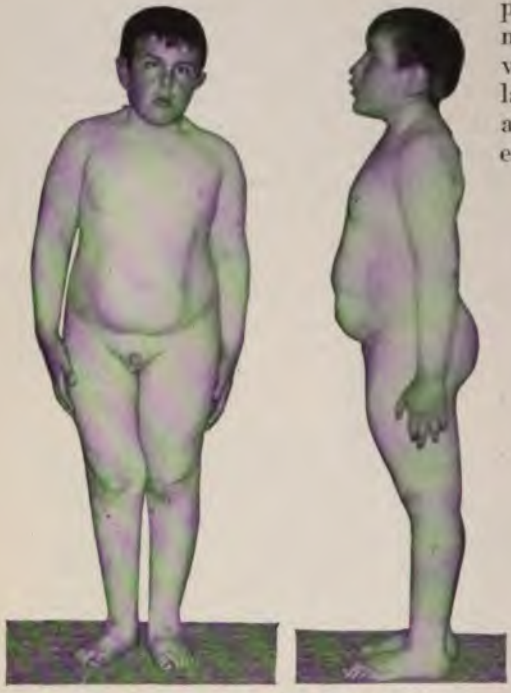
[1] "Neurol. Centralbl.," July 1, 1901.

underlying muscular defects and maintain a fictitious appearance of muscular development that is belied by great weakness. In the same way the muscles presenting pseudohypertrophy look immensely strong, and feel very firm, but are found lacking in contractile power and subject to fibroid shortening.



Figs. 164 and 165.—Progressive amyotrophy masked by fat except in legs and face.

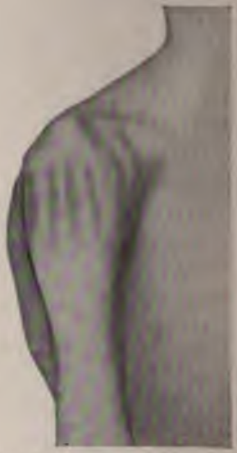
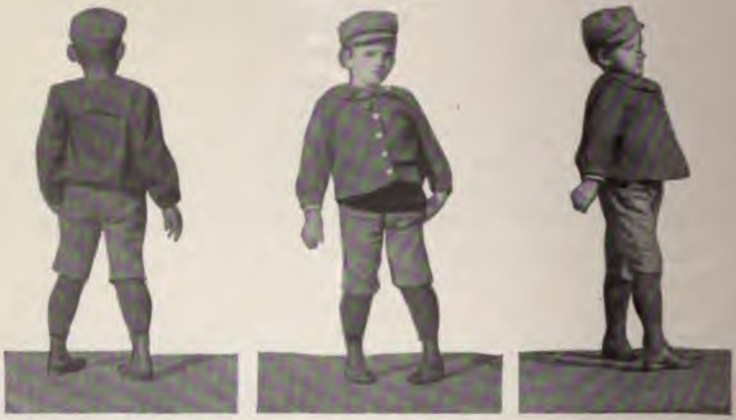


Fig. 166.—Showing the deltoid in advanced myopathy.



Figs. 167, 168, and 169.—Waddling gait in a case of pseudohypertrophic paralysis.

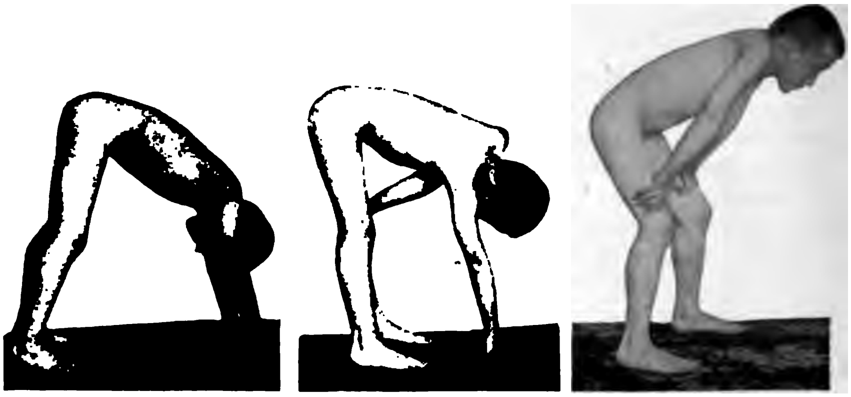
The *attitude* presented by the patients is the result of the atrophic and paretic condition of the affected muscles and of the contractures that sometimes appear in them. It, therefore, varies with the location of the disease. Owing to the shortening of the calf muscles, little difficulty is found in maintaining the ankle-joint at a proper angle unless an equine position of the foot has developed, causing the patients to stand upon their toes. Standing then becomes extremely difficult. The position at the knee and hip is often entirely dependent upon the ligamentous support of these joints. This allows the pelvis to tip forward upon the head of the femurs and necessitates a strong anterior spinal bend to carry the head and shoulders back into the line of gravity for the maintenance of the equilibrium. The prominence of the abdomen is thus produced and is accentuated in turn by the weakness of the abdominal muscles. In some cases the head is tilted backward, but when the spinal curve is pronounced the chin may rest on the sternum. Ordinarily the feet are kept wide apart to increase the base of support.

The *gait* is broad-based and waddling (Figs. 167, 168, and 169). The foot to be advanced is clumsily put forward, the pelvis tilting sharply downward on that side, the body being inclined over the supporting foot as a counterpoise to the swinging limb. This is repeated in the opposite sense at the next step, and the patient advances in a swaying, waddling, awkward fashion, stumbling over the least obstacle, and falling heavily if the very unstable equilibrium is momentarily lost. Mounting stairs is often an early difficulty.

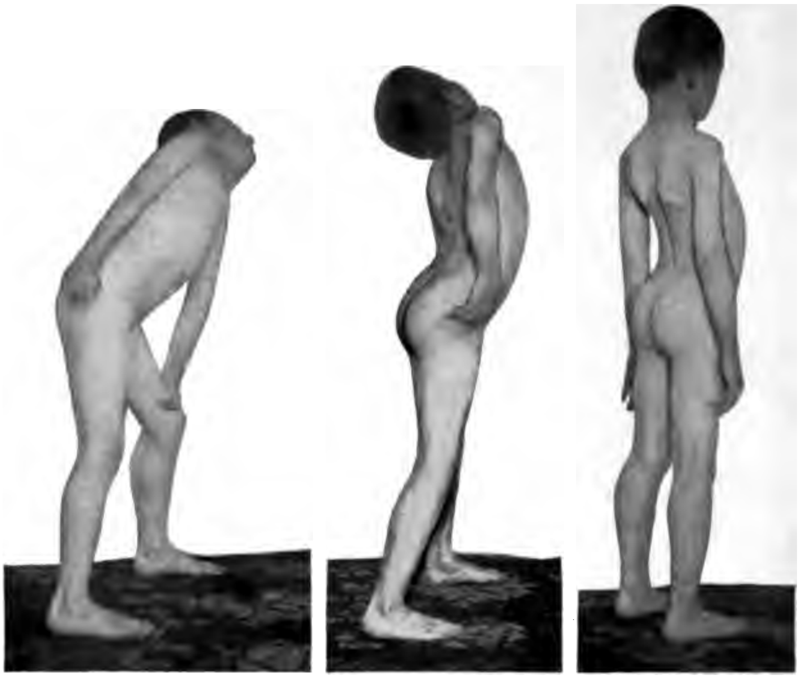
The manner of rising from the ground is most characteristic. If placed on the back, the patient may have great difficulty in rising at all. His usual plan is to turn over on his face, then huddle himself together, and get upon his knees. The usually weakened psoas, vastus, and gluteal muscles are now inadequate to the effort of lifting him. He, therefore, advances the body into the "all fours" position, and, carrying the weight of the head and shoulders on the arms, pushes up the lower end of the trunk with his legs as a cow gets up by the hindquarters. The hands are now brought toward the feet, one is placed above the knee on the same side, then the other at a higher point on the thigh of its own side; the shoulders and head are pushed upward, the pelvis tilts forward, the sway-back suddenly appears, and the patient attains the erect attitude by a process of climbing up his own legs (Figs. 170 to 175). In late stages standing and walking may be impossible.

It is needless to say that in the cases in which the legs and pelvic muscles escape or are only slightly affected, the attitude, gait, and manner of rising are not disturbed. The sway-back may also disappear when the patient is sitting, or may then give place to a rounding of the back, the patient resting his elbows on his knees or otherwise gaining a fictitious support for the upper part of the trunk.

The *upper extremity* is most affected by the impairment of the muscles of the shoulder-girdle. Next in frequency the brachial group is affected, while the muscles of the forearm and hand are usually spared. In the *lower extremity* wasting rarely avoids the gluteal and psoas groups and commonly affects the anterior crural distribution. The calf-muscles with the glutei furnish favorite locations for pseudohypertrophy, while the calf-muscles in addition are the commonest of all locations for fibroid contractures, which may, however, appear in any muscle in the late stages of atrophic shrinking. The small muscles of the foot may escape. The peronei are selected first in the leg type.



Figs. 170, 171, and 172.—Method of rising from the ground in cases of myopathy.



Figs. 173, 174, and 175.—Method of rising from the ground in myopathy.

The *tendon-reflexes* are usually diminished, and when wasting or pseudohypertrophy is apparent they are commonly abolished. The ordinary case shows only a quantitative reduction of *electrical* muscular *stimulatability* for both currents. In rare instances, and usually in the peroneal or so-called neurotic type, the reaction of degeneration may be obtained. *Fibrillary twitchings* and *severe pains* are equally rare, but are found under similar circumstances. The *sphincters* are not affected. *Sensation* is intact in all its modes and tenses. A large proportion of these cases, and perhaps all of them dating from early childhood, show some *mental backwardness* and apathy. Trophic disturbances are not encountered, but *growth* may be retarded and the bones may be diminutive.[1]

Course.—These conditions are progressive. The muscular wasting usually steadily advances with its attendant and increasing weakness. Some cases present stationary periods without assignable cause, and resume their course without adequate explanation. As a rule, the younger the age at which the disease appears, the more rapidly does it incapacitate the individual. It commonly occurs in girls at a later period than in boys, and then runs a slower course. When commencing in childhood, sometimes an age of forty years may be reached, but death usually occurs between twenty and thirty. Not, as a rule, fatal in itself, except by pneumogastric accidents, death ordinarily occurs from intercurrent disease.

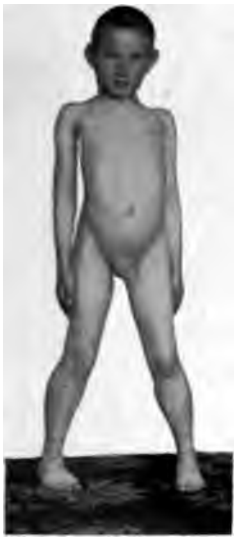


Fig. 176.—Showing attitude, facies, deltoid, and calf deformities in a case of pseudohypertrophic paralysis.

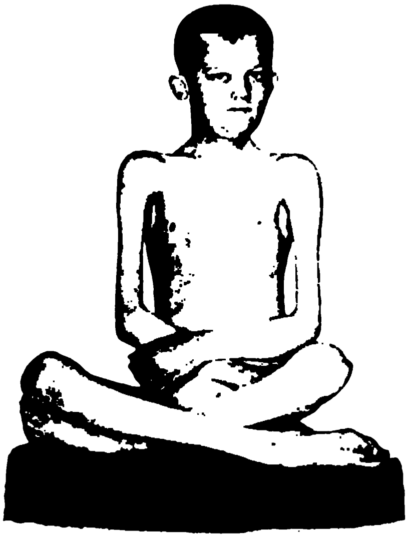


Fig. 177.—Advanced case of progressive myopathy, at one time marked by pseudohypertrophies. Observe facies, deltoid conformation, and comparative integrity of hand and forearm.

Varieties.—A number of clinical varieties have been described and are worthy of some attention, but it should be borne in mind that they are not distinct morbid conditions.

The earliest recognized form is the one called *pseudohypertrophic paralysis.* It usually appears in early childhood, affects boys much more frequently than girls, and is marked by extreme enlargement of the calves and buttocks. These stand out in intensified relief next the wasted thighs and forward-tilted pelvis. The psoas group is early affected, rendering going up-stairs difficult at an early date. False hypertrophy is also commonly found on the dorsum of the scapula, in the lower border of the pectoralis, and the lower part of the deltoids. The serratus magnus, erector spinæ, and humeral muscles commonly suffer early. The myopathic facies is not well marked, but can be detected in late cases. The forearms, hands, and feet are commonly spared until the last. The enlarged muscles finally shrink, and present all the features of those that shrink from the first.

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[1] Schultze, "Deutsch. Zeit. f. Nervenh.," April, 1899.

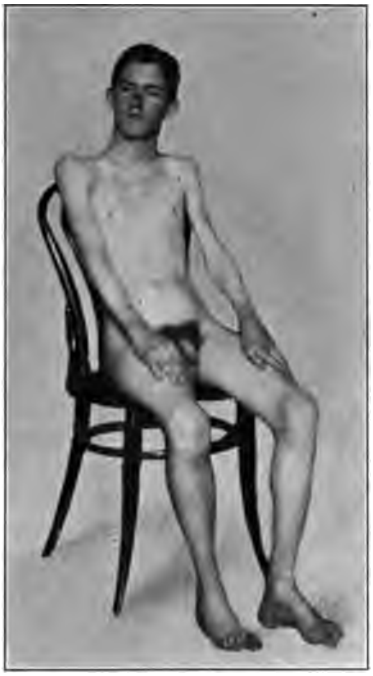

Fig. 178.—A mixed case showing facioscapulohumeral involvement, peroneal wasting, and the foot deformity of Friedreich's disease. There were fibrillary twitchings and the reaction of degeneration in the leg muscles.

The *peroneal* or *leg variety*, sometimes called the *Charcot-Marie type*, or the neurotic form, is marked by the early atrophic shrinking of the peroneal muscles. The calves are next involved and the thighs follow. The disease may thus be confined to the lower extremities for years. Eventually it invades the trunk and upper extremities, first affecting the hands. It shows fibrillar twitchings, the electrical changes of degeneration, and presents undoubted cord-lesions.

The *juvenile type of Erb*, also called the brachial form, affects mainly the muscles of the arms and shoulders, and appears in early youth, and usually in several members of the same family.

The *facioscapulohumeral* form, or the *type of Landouzy-Déjérine*, affects face, shoulder, and arm, or may be considered as the brachial form plus facial involvement. These cases furnish marked instances of the myopathic facies.

Several of these forms may be present in the same patient, as the leg type with the facioscapulohumeral variety, or pseudohypertrophy in the legs and the brachial type above. The hypertrophic variety regularly becomes shrunken finally, and lumps of false hypertrophy, on the other hand, may appear in muscular groups otherwise greatly wasted.

Prognosis.—The outlook is always unfavorable, but some of these amyotrophies appear late in life, and do not apparently shorten it. The menace of the disease falls upon the respiratory apparatus and heart. Intercurrent diseases also find an easy prey in the weakened organism.

Treatment.—Unfortunately, all forms of medicinal treatment have given negative results. Carefully selected exercises, graduated to the capacity of the weakened muscles, promise the most good. Gowers early insisted upon this, and Wiener[1] has recorded a very encouraging instance in which much improvement was secured. Massage to the wasting muscles may have a beneficial effect. The application of electricity is usually rendered impossible by the painfulness of the currents required to actuate the muscles if much wasted, and it is not certain that its vigorous use is harmless. General measures pertaining to the health, comfort, and education of these unfortunates will be suggested by their individual requirements. Sometimes a tenotomy of the heel-tendon is required to keep them on their feet, but the use of sustaining corsets and braces usually augments the muscular feebleness by depriving the parts of their only natural exercise.

ARTHRITIC MUSCULAR ATROPHY.

All varieties of joint-disease may cause wasting of the muscles related to the joint. This occurs in two ways: (1) The inflammation may involve nerve-trunks passing the joint, set up a neuritis and produce motor, sensory, and trophic disturbances in the distal distribution of the affected nerves below the diseased joint. The process is simply a neuritis. (2) The articular filaments may be disturbed by the arthritis. Irritation ascends to the spinal centers and disturbs the trophic control of those cells related to the muscles physiologically associated with the joint and located on the proximal side of the affected articulation. It is a process acting through the reflex arc. We are to consider the second variety only. It is placed in this connection because it is a disease marked by disturbance of the spinal gray matter.

Etiology.—Any joint-lesion involving the articular filaments is competent to set up an arthritic muscular atrophy. It may thus follow simple, acute, or chronic arthritis, infectious arthritis, the arthritis of gonorrhea, of rheumatism, or of traumatism. It has no proportionate relation to the severity of the joint-disease, but rather appears to be relative to the irritant qualities of the articular mischief.

Symptoms.—In acute joint-lesions the muscular atrophy usually begins within a month, sometimes within a week, and the muscles promptly show diminished bulk and altered contours. In chronic arthritis—for instance, in gout and chronic rheumatism—it may very gradually develop, appearing very insidiously and requiring many months for its complete evolution. The striking peculiarity of the atrophy is that it is usually, and at first always, confined to the *extensors* of the joint, and involves them in their entire length. The only exception is in arthritis of the ankle-joint, in which case the calf-muscles waste. Arthritis of the shoulder affects the deltoid, of the elbow the triceps, of the wrist the extensors, of the fingers the interossei, of the hip the glutei, of the knee the anterior crural muscles, of the ankle the calf-muscles, of the toes the interossei of the foot. When the flexors are also involved, they suffer to a less degree. *Sensory* disturbances are absent, or are such as are referable to the arthritis. In occasional instances the joint-inflammation also affects the nerve-trunks in its neighborhood, and a neuritis is added, with symptoms below the joints. The wasting involves the affected muscles uniformly from end to end, and there is a corresponding *loss of power*. The *electrical excitability* of the muscles may be reduced slightly, but is generally increased relatively to the bulk of muscle present. The reaction of degeneration is never found. The *tendon reflexes* are exalted. In very pronounced cases a rectus-clonus or an ankle-clonus may be obtained. When the joint-disease subsides, the tendency is for the muscles to slowly recover, but in quite a proportion of cases they never completely regain their former vigor, and sometimes they remain permanently wasted.

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[1] "American Journal of Med. Sci.," Oct., 1896.

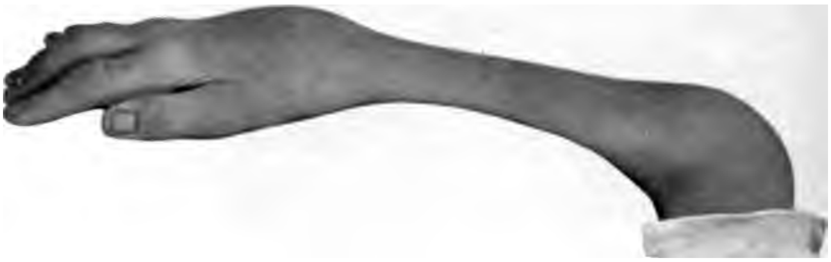

Fig. 179.—Severe arthritic muscular atrophy in a case of multiple arthritis.

Morbid Anatomy.—The atrophic muscles are flaccid, pale, and inelastic. The fibers are simply diminished in size and there may be a little interstitial fibrosis. The only change in the nervous apparatus thus far recorded is in the nerve-terminals within the inflamed joint, due to their implication by the local disease. Better technic may be expected to discover cellular cord-changes.

Pathology.—Paget, J. K. Mitchell, Vulpian, and Charcot early recognized the reflex mechanism of articular muscular atrophy. Valtat found that arthritis experimentally produced was followed by the limited atrophy in question, and Raymond found that this atrophy did not follow if the corresponding posterior nerve-roots were divided. Hoffa clinched the matter by irritating joints on both sides and cutting the nerve-roots on one side. The atrophy only occurred on the side retaining an unbroken reflex arc. The anatomical rule is that nerves supplying the extensors of a joint also innervate the joint. The physiological association of arthritis and atrophy limited to the joint-extensors would in itself indicate the spinal element in the pathological mechanism. An interesting question is whether the symmetrical joint-changes and osteal conditions of gout and chronic rheumatism are not primarily due to the action of toxic substances upon the spinal cells governing the nutrition of bones and joints. Some cases of panarthritis or multiple arthritis symmetrically distributed, as well as the distortions of arthritis deformans and gout, strongly suggest a central disturbance as the principal and initial factor.

The diagnosis of uncomplicated arthritic muscular atrophy is usually easy. It depends: (1) On the limitation or excess of atrophy in the extensors of a joint actively diseased or formerly arthritic; (2) upon the quantitative electrical changes and absence of the reaction of degeneration, and (3) upon the increase of myotatic irritability. A microscopic section of the muscle would show a simple diminution of the muscle-fibers. When the joint-inflammation invades adjoining nerve-trunks, a neuritis may at the same time give symptoms below the affected joint. The atrophy usually remains sharply limited, in strong contrast to the invading tendencies of progressive forms.

The prognosis depends upon the joint-condition. If the arthritis subsides, the atrophy usually disappears. This is the rule in acute arthritis with early recovery. In more persistent lesions the atrophy is of corresponding duration and less likely to disappear when the joint recovers.

The treatment is primarily of the joint and secondarily of the muscles, by massage, electricity, and gentle exercises.

CHAPTER IV.

LESIONS PRINCIPALLY AFFECTING THE WHITE MATTER OF THE CORD.

THERE are a number of diseases of the spinal cord in which the lesions are principally confined to certain white tracts, and they are frequently classed among the so-called system diseases or diseases of systematized lesions. The conception on which such a classification was based has been so much modified that it seems better to drop it altogether, especially as there has always been a great divergence of opinion as to what should be embraced under that caption. Acute poliomyelitis, for example, which was one of the usually accepted types, is, as we have seen, the result of accidental infection by way of the vascular supply. Posterolateral sclerosis, or ataxic paraplegia, is also usually based upon anatomical vascular conditions, and presents so many variations that it can not be considered as systematized from any point of view. In locomotor ataxia the spinal lesion is as sharply delimited as in any other, but it is a disputed question whether or not the cord-lesion is secondary to initial root-changes. The morbid anatomy of the disease is so widespread that to class it as a systemic cord-lesion is to seriously contract the full view of the disease, and to ignore its neuritic and cerebral features. The secondary degenerations of the cord-tracts have been considered in connection with the various cerebral and spinal lesions which give rise to them. Primary spastic paraplegia, or Little's disease, has been discussed with the Cerebral Palsies (see p. 242), as it is dependent upon a congenitally unfinished condition of the upper neurons, and furnishes one of the diplegias of childhood.

Because the spinal symptoms of locomotor ataxia are the most salient of its many manifestations, and as its differential diagnosis is a constantly recurring problem in cord diseases, it is taken up at this time. In fact, however, it is a disease of the entire nervous system and must properly be so classed. The student should never lose sight of the fact that the cord-lesion in tabes dorsalis is only a small portion of its morbid anatomy.

TABES DORSALIS: PROGRESSIVE LOCOMOTOR ATAXIA.

Tabes dorsalis, progressive locomotor ataxia, leukomyelitis posterior, sclerosis of the posterior columns, are some of the many names applied to a symptom-group of vast proportions. It is a disease almost always preceded by a syphilitic infection, usually marked by a degenerative process in the sensory nerves, posterior nerve-roots, posterior columns of the spinal cord, and often by similar changes in the medulla and cerebrum. Clinically, there is commonly disturbance of cutaneous and joint sensation; impairment of the muscular sense; incoördination of muscular movements, especially in the lower extremities; diminished or abolished knee reflexes and lessened pupillary reflexes to light; visceral and trophic disturbances and paroxysmal attacks of pain. The disease presents a progressive tendency toward complete helplessness.

In the description of this protean disease the masterly lectures of Marie [1] have been found of great help, and in some measure his arrangement of the subject is followed herein.

Etiology.—The principal factor in the etiology of tabes dorsalis is syphilis. It is impossible at present to say that it is an indispensable element in a given case, because chronic intoxication with ergot may induce tabetic features and similar degenerations. The same changes are found in pellagra, and it is at least supposable that other toxic states may lead to tabetic conditions. Since attention was first called to the parasyphilitic nature of locomotor ataxia by Fournier, in 1875, statistics on the subject have shown an increasing proportion of cases presenting syphilitic antecedents. In 1894 [2] he states that in the examination of 750 cases he finds the percentage of syphilitics varying from 87 to 93 for each hundred, taken separately. Erb reported 89 per cent., Déjérine, 92 to 94 per cent., Sachs over 90 per cent., Peterson, 71 per cent., and the author, in the last 100 consecutive cases occurring in private practice, has failed to find history or evidence of luetic infection but twice. It is safe to say that, practically, 9 out of 10 cases are syphilitic, and that the tenth case is open to very serious doubts if it occurs under ordinary circumstances of life. Reports of cases of tabes acquiring syphilis subsequent to the development of ataxia can not be obtained. Such immunity argues prior infection.

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[1] "Leçons sur les Maladies de la Moelle," Paris, 1892.

[2] "Les affectiones parasyphilitiques," Paris, 1894.

The tabetic symptoms may appear in from one to thirty-five years after the initial syphilitic sore, but ordinarily develop from the fifth to the fifteenth year. It is a noticeable and most important fact that the apparently mild cases of syphilis—those in which the secondary features are indistinct or entirely lacking—loom largely in the histories of tabetic patients. These are precisely the cases in which an intense medication is not urged or in which faithful pursuit of it by the patient can not be secured. It must be said, however, that cases of tabes are only too frequently encountered in which syphilis has run a severe course and in which persistent and intensive medication has been heroically prescribed and faithfully borne for two and three years, and yet tabes has subsequently developed.

It is rare for tabes to develop before the *age* of twenty-five years, just as it is rare for syphilis to be acquired before adolescence. In all cases appearing in childhood, hereditary syphilis is present. The great proportion of cases appear between thirty and forty-five. As a rule, the alleged etiological factors of tabes, other than syphilis, are the very ones which contribute to the frequency of syphilis. Thus, the male *sex* is about ten times as frequently affected with tabes as the female. The same proportion obtains for syphilis. But among females subject to tabes Erb found the percentage of syphilitics to be 89.5 per cent., practically the same as in males.

The *race* question tells the same story. In rural communities and among the orthodox Jews cases of syphilis are comparatively rare, and tabes equally infrequent. Excessive *venery* has been accused of producing tabes. Its relation, if it has any, is by the increased exposure to luetic infection it entails. The action of *cold, rheumatism, overexertion, alcoholism*, and *acute fevers* has been much insisted upon in former years, but we know nothing definite about them. Many cases attributed to *traumatism* have been misinterpreted cases of organic indiscriminate or combined cord-lesions. In other instances the accident leading to the injury has been the result of tabes, not its cause, as in falls and fractures, the result of the previously unrecognized incoördination. The question, however, is still debatable. Prince,[1] after a critical examination of the cases in literature, is inclined to deny the traumatic origin of tabes. Schittenholm,[2] after reviewing the subject and the literature, reaches the conclusion that trauma as a unique cause of tabes is not proven, but that it may aggravate the tabetic condition.

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[1] "Jour. Nerv. and Ment. Dis.," Feb., 1895.

[2] "Münch. med. Woch.," 1903.

A *neuropathic heredity* appears to play a predisposing part to some extent. We thus not infrequently encounter epilepsy, hysteria, chorea, insanity, hemiplegia, and diabetes in the ascendants of tabetics, and even, though most rarely, other cases of locomotor ataxia. May it not be that such instances indicate an inherited vulnerability on the part of a certain portion of the nervous apparatus to the toxic effects of luetic infection? It must be evident that only a very small percentage of syphilitics develops tabes.

The bearing of *occupation* is that related to syphilis. Artists, actors, journalists, and soldiers are frequently affected, physicians and surgeons not infrequently, clergymen most rarely. Country laborers, so greatly exposed to traumatism and exposure, very seldom present tabes or syphilis.

In spite of the immense importance of syphilis in the causation of tabes dorsalis, it must be kept in mind that the nerve-lesion is not comparable to the tertiary or secondary specific lesions, and is not amenable to antiluetic remedies. It is a degeneration showing progressive sclerotic changes that are beyond repair. Following the views of Strümpell and Marie, we may hypothetically attribute the ascending degenerations in the nerves, roots, spinal cord, and brain to the action of a syphilitic toxin which primarily affects the cell-bodies of the fibers making up the afferent tracts.

Edinger undertakes to explain the apparently selective activity of the toxic agent upon certain physiological nervous tracts and structures by an ingenious hypothesis. Those nerve-elements which are the most constantly employed are most affected. Thus, the sensory tracts, especially in the lower parts of the cord, the pupillary activities, the bladder function, and the intestinal field are early and commonly affected. The hypothesis conjoins the elements of (1) fatigue locally precipitating the effects of (2) a general toxic state.

The theories as to the pathogenesis of tabes are numerous and varied. Four principal ones may be mentioned. First, the toxemic theory with syphilis as the usual origin of the toxin. Second, the strangulation theory of Obersteiner and others who find the initial factor in pial thickening, whence arises the posterior root and cord changes. Third, Marie's recent contention that the changes start in a lymphangitis of the posterior columns of the cord. And, fourth, the "Ersatz" theory of Edinger above indicated. Orr and Rowe,[1] while granting a hereditary or acquired weakness of the nervous apparatus, believe that the process is located in the sensory neurons of the posterior cord tracts because of the anatomical absence of the neurilemma at the point where the sensory root fibers enter the cord. They find this to be the initial point for the degenerative process.

Morbid Anatomy.—In this section it is purposed to take up the morbid anatomy of the nervous apparatus in tabes dorsalis. The morbid anatomy of the trophic lesions in the bones, joints, and soft structures will be described respectively with their clinical features. As the disease is essentially marked by ascending degenerations, it is well to commence at the periphery.

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[1] "Brain," Winter, 1904.

The *spinal nerves* are usually found to present a degeneration which is greatest at the periphery and most marked in, if not entirely confined to, the sensory fibers for the skin, muscles, and joints. The trophic centers for these fibers are in the posterior root-ganglion in part, but it seems probable that there are also superficial peripheral trophic cells for at least a few of them. The *muscle spindles* which undoubtedly have a sensory function are constantly found degenerated. By some writers these nerve-changes are denominated a neuritis, but histologically the changes conform to a Wallerian degeneration. The *spinal nerve-roots* present a marked difference between the anterior or motor members, which are ordinarily normal, and the posterior sensory roots, which are usually, if not invariably, greatly degenerated. Only when changes have taken place in the anterior cornual cells do we find changes in the anterior roots, and then there is corresponding amyotrophy. This is

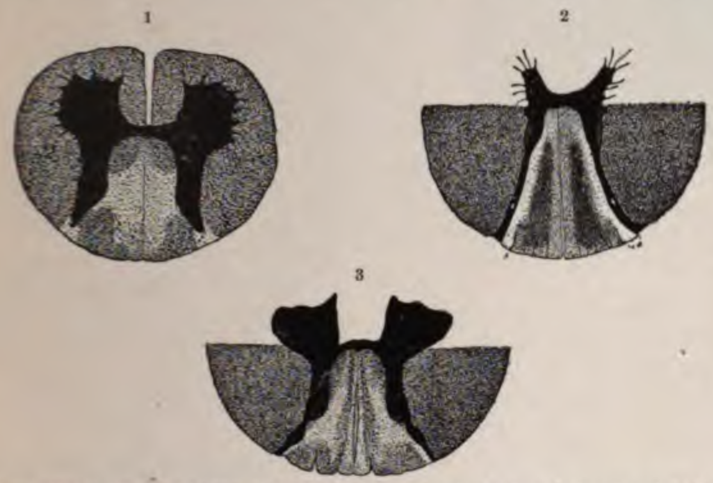

Fig. 180.—Sections of the cord in an early case of tabes. 1, Lumbar region; 2, dorsal region; 3, cervical region (Marie).

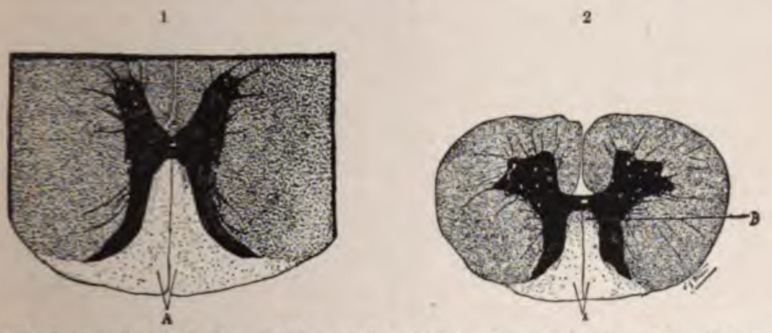

Fig. 181.—Section of cords in advanced tabes. 1, Dorsal region; 2, cervical region. Sclerosis shown by white region (Marie).

usually a late and secondary or accidental feature in tabes. The *posterior roots* and their ganglia show much disease. Oppenheim, Simmerling, Marie, Obersteiner,[1] Juliusberger and Meyer,[2] describe changes in the *root-ganglion* cells, which are found reduced in size and number. Nuclear displacement and granular changes were demonstrated by Nissl's method, according to the last-mentioned authors. The fibers within the ganglia were also atrophic. The posterior roots are practically always degenerated. The posterior root-ganglion occupies, therefore, a prominent position in the development of tabetic lesions, as it exercises a trophic control, both downward over the sensory fibers of the nerves and upward over the posterior root-fibers and their continuation within the posterior tracts of the cord. Changes in the visceral branches of the *sympathetic nerves* of a degenerative character have also been reported, and degeneration of the fine fibers of the sympathetic arising from the viscera and entering the cord by the posterior roots has been demonstrated in a series of cases.[3]

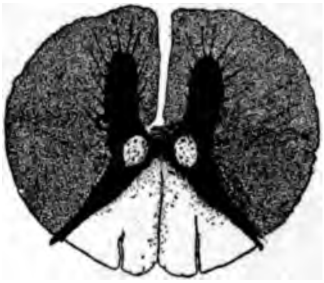

Fig. 182.—Section in lower dorsal cord in a case of advanced tabes showing complete sclerosis of the posterior columns and of Clarke's cellular columns (Marie).

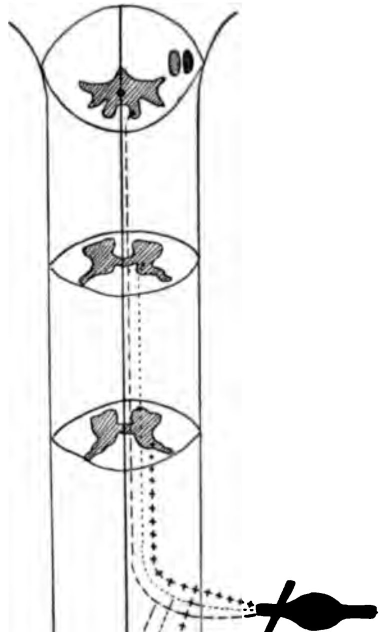

Fig. 183.—Schematic representation of the courses pursued by the sensory root-fibers entering the cord from the root-ganglion (Marie).

To understand the distribution of the *cord-lesions* it is necessary to recall that the posterior root furnishes three sets of fibers, which enter the posterior horn by different routes and at different levels. The fibers of the *first group*, almost as soon as they reach the cord, enter the posterior gray to the inner side of the horn at its posterior third by a short route through the tract of Lissauer. Those of the *second group* pass to the inner side of the first in Burdach's column, and enter the middle third of the horn's inner border at a level considerably above their point of entrance to the cord. Some of them reach Clarke's column. The *third group*, arising mainly from the lower limbs, enter still nearer the median line and pass up nearly the entire length of the cord, constituting the columns of Goll, and terminate in the bulbar gray. The tract of Lissauer lying between the head of the posterior horn and the periphery of the cord among the entering posterior-root fibers, is made up of fine fibers arising from the posterior roots either as collaterals or as direct continuations of the numerous finer fibers of the root. These fibers of Lissauer's tract, after ascending a short distance, enter the gelatinous substance of Rolando, which caps the posterior horn, and some of them penetrate the posterior horn itself. This tract also degenerates in tabes.

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[1] "Berlin. klin. Wochens.," Oct. 18, 1897. [2] "Neurolog. Centralbl.," Feb. 15, 1898.

[3] Roux, "Lesions of the Sympathetic System in Tabes," Paris, 1900.

Embryologically, the posterior columns of the cord, including Lissauer's tracts, are an accession to the cord. They arise in the lateral neural plaques, which are finally represented by the posterior-root ganglia, and enter the cord by way of the posterior roots, pursuing their course upward in the manner indicated. This part of the cord only reaches full development at birth. It is to this posterior, exogenous, independently developed portion of the cord-structure that the spinal lesions of tabes are principally confined.

These vary at different levels and at different stages of the disease. In long-standing cases the entire portion of the cord embraced between the posterior horns and the commissure may be sclerosed from the filum terminale to the medulla. In incipient cases the tracts of Lissauer and the columns of Burdach are affected at the lowest level, the columns of Goll above. Ordinarily, the sclerotic changes of tabes are most marked in the lumbar cord, where they commonly begin; in other cases the cervical enlargement is most affected; again, in others, the intracranial lesions of the medulla predominate, and when associated with cortical changes we have the progressive paralysis of the insane. Clinical varieties correspond to these various preponderating locations of the sclerotic process, and all intermediate varieties and combinations of lesions and clinical types are encountered. This sclerotic change, like that in the nerves, is a pure Wallerian degeneration.

The lesions of the *spinal gray* in tabes, as far as now describable, consist of a degeneration of the fine nervous reticulum about the cells of Clarke's column near the inner border of the neck of the posterior horns. The change is an early one and most marked in the outer cells of the group. This reticulum is made up of the terminal brushes of fibers from the posterior roots. Ordinarily, the cells themselves are spared, but in some cases they, too, show degenerative changes, and the *direct cerebellar tracts* and *Gower's ascending anterolateral tracts* are then also sclerotic. These two cord-tracts find their trophic supply in the cells of Clarke's column; hence their degeneration when these cells are affected. Clarke's column begins in the upper lumbar segments and extends to the seventh or eighth dorsal, when it becomes extremely attenuated and practically disappears at the second dorsal, to again appear above the cervical enlargement in the upper cervical cord. The lesion of Clarke's column is, therefore, most marked in the lower dorsal region.

The posterior horn proper, according to Lissauer and others, shows only insignificant changes except in the caput of Rolando, where the fine fibers and radiating fibers from the posterior roots are customarily degenerated.

The cord-lesions in tabes are commonly symmetrical, but not infrequently in early stages one side will show more sclerosis than the other, often corresponding to greater symptoms in the limb or limbs of that side.

In addition, it is to be noted that there is a *clouding of the meninges* overlying the sclerotic tracts in the spinal cord, to which much importance has been given by some who see in it a *meningitis* forming an initial pathological process in tabes. This and the cord-changes can be frequently recognized by the eye, but in incipient cases recourse to the microscope is required to decipher the morbid condition. In some instances the meningeal condition reaches one of chronic inflammation, which, in turn, may cause a *marginal myelitis* that may even extend to the pyramidal tracts.

Bearing upon the question of meningitis is the fact that the *spinal fluid* during life commonly presents a lymphocytosis, as has been abundantly proven by Widal, Sicard, Ravaux, and Schoenbrun.

The *cranial nerves* are all liable to degeneration, but this tendency is most marked in the optic and auditory nerves, which in effect are cerebral lobes. Changes of a similar sort are not rarely found in both roots of the trifacial or in the nuclei of the motor nerves of the eyeball and of the intra-ocular muscles, in the glossopharyngeal, pneumogastric, facial, and hypoglossal. Involvement of the iridociliary apparatus is, perhaps, the most common of all.

The *cerebral lesions* of tabes, aside from those of the cranial-nerve nuclei or even embracing them, are those of *paretic dementia*, the description of which is contained in the second part of this book. With this disease tabes has the most intimate relations. Fournier has gone to the point of considering them of identical nature and only varying in the primary location of the lesions. The parasyphilitic theory of paretic dementia rests on exactly the same sort of a foundation as does that of tabes dorsalis. A certain proportion of cases of tabes develop paralytic dementia; a large number of paretic dements present tabetic symptoms and tabetic cord-lesions. Both diseases commonly have identical bulbar and cranial-nerve symptoms and lesions. Though Jendrassik tends to consider the cerebral lesions of tabes as always initial, they are probably concomitant only, and due to the same specific cause. Their association in point of time and development is open to all possible modifications throughout all the varieties of each affection. The most pronounced types of both syndromes are presented as the sclerotic process falls first and most severely, respectively, on the caudal or cephalic extremity of the cerebrospinal system.

Symptoms.—From the extent of the morbid anatomical changes in tabes it must be evident that its symptomatology embraces nearly every bodily organ and function. The clinical features of the disease can only be fully presented by taking them up systematically. Afterward an attempt will be made to group them in describing the common clinical varieties of the malady. As we proceed, the relation of the various symptoms to the course of the disease will be indicated. Their relative frequency will be shown in tabular form.

Motor Disturbances.—The motor disturbances of tabes, while not the earliest, are among the most important, and are the ones which usually first seriously attract the patient's attention. They consist of: (1) *ataxia ;* (2) *involuntary movements,* and (3) *palsies.*

Tabetics deprived of their muscular sense by the posterior sclerosis or by the degeneration of the peripheral sensory fibers in the muscles and joints can not determine the exact positions of their limbs without the aid of vision. This is, no doubt, increased to some extent if attended by cutaneous dysesthesia, which is frequently present. They thus "lose their legs in the bed" in marked cases, and have to verify the position of their limbs with their hands or eyes. In less pronounced cases they can not with closed eyes duplicate with one limb the position passively given the other by the examiner, or do so with uncertainty. Another manifestation of the perturbation of the muscular sense is an inability to accurately distinguish the difference in weight of similarly shaped objects held in the hands. The normal individual can usually determine a difference of five per cent. This symptom can sometimes be shown by having the patient first write a short sentence with open, and then repeat it with closed, eyes. The uncertainty of position and the consequent incoördination is then sometimes graphically demonstrated, while grosser movements with the upper extremities may be tolerably exact. Many patients fail to touch the nose or ear or any given point with the index-finger when the eyes are closed.

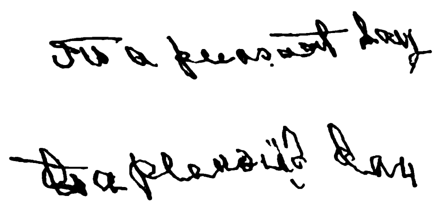

Fig. 184.—Ataxia of the upper extremities shown in the handwriting. 1, Written with eyes open; 2, with closed eyes.

Tabetics commonly present the *sign of Romberg* early if the lower portion of the cord is involved. With closed eyes they sway or may fall heavily if the feet are placed close together, and may even be unable to stand with a broad base of support. Walking backward with closed eyes will almost invariably determine incoördination in the lower extremities if present in the slightest degree. So will attempts to stand on one foot. In advanced cases this loss of muscular sense causes so marked an ataxia that walking is impossible, even with the eyes open.

To this source we must also attribute the *ataxic gait* that is so characteristic when well developed. It by no means appears clearly in all cases, but may usually be induced to some degree by having the patient walk with closed eyes, or may be manifest in the difficulty of executing a prompt "about face," in suddenly rising from a chair and attempting to immediately walk forward, in descending a stair, in halting suddenly when advancing, and in marking time after the military fashion. In crossing one knee over the other the moving limb is often raised too high and too vigorously dropped across its fellow. When the gait is plainly ataxic, the patient rises from the chair with some suddenness, as if lifted by a spring, then balances a moment before putting himself in motion. The first steps are usually taken briskly and the patient may even plunge forward. The legs are thrown out widely. The foot goes forward irregularly with some rigidity, the toe up, often departing laterally from the direct line, and is brought down with a shock upon the heel, or flat-footed. As the body is advanced, there is a tendency to overextension of the supporting knee-joint, which often produces a backward bowing of the limb and may constitute a marked deformity.

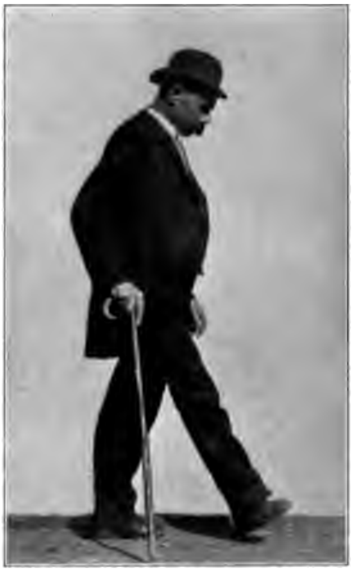

Fig. 185.—Gait in tabes. Observe overextension of supporting knee, rigidity of advancing leg, elevated toe, heavily descending heel, watchfulness of steps, and assistance by cane.

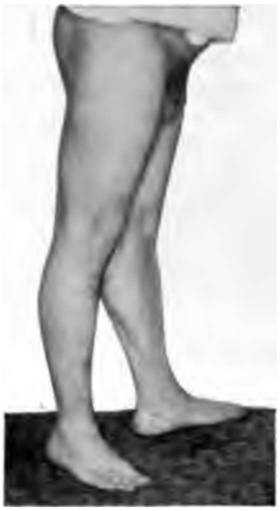

Fig. 186.—Showing the tendency to overextension of the knee-joints in tabes, to secure firmness in standing.

The foot is usually raised too high at the end of the step, but is also prone to catch upon any trifling obstacle as it swings forward. The opposite foot is then advanced in a similar irregular way, and the patient almost invariably aids himself by carefully watching his feet and by the additional assistance of one or two canes (Fig. 185). In some cases there is considerable uniformity in the manner of taking each step, but as often no two paces are exactly alike, and the patient, instead of advancing in a straight line, swerves more or less to one and the other side, often bumping into neighboring objects.

When he sits down he does so with suddenness, as if both knees gave way too soon, as they often do. In the same way he can not gradually lower himself from the erect position to a squatting attitude, but drops suddenly when the knees are partially bent. The sudden giving way of a knee sometimes causes him to fall, and, as a rule, his attitude and gait denote every effort to trust these joints as little as possible and to fix them by overextension.

When the *upper extremities* are markedly ataxic, it is shown in the manner of grasping a small article, toward which the hand goes with wide-spread fingers, and grabs down on it as if it might escape by flight. Finally, walking becomes impossible, and the upper extremities may become useless except for wide and inexact movements. Commonly incoördination is most marked in the lower extremities, but in the cervical type of tabes the condition may be more pronounced in the arms. In all these motor affections, however, muscular power is not necessarily impaired. It is only the muscular sense that fails. The ataxia is commonly of insidious development, but in some instances comes on abruptly, usually as the result of some physical strain or exhausting condition or after a period of disuse of the limbs, as, for instance, after an illness or fracture. In cases where it is slowly increasing it may show sudden intensification, some of which may recede, but rarely does such sudden increase of ataxia fully pass away.

Involuntary movements in tabes dorsalis are not uncommon, and probably more frequent than reports indicate, as they are obscured by the major features due to incoördination. In the early as in the late stages they may be observed in some cases, and usually are uniform for the given patient. They may consist of a sudden involuntary movement of the thumb or a finger, or the turning of the wrist, or the jerking of the arm or foot. In a case now under observation the entire lower extremity is frequently violently jerked, mainly by the flexors of the hip, and these movements may be repeated rapidly, but irregularly, for several minutes, and even for an hour or two daily, irrespective of fatigue or position. Such jerkings of the legs are prone to occur during sleep. In exceptional cases and in their terminal stages both lower extremities may be forcibly drawn up to the body. This occurs upon coughing, sneezing, or sometimes upon voluntary efforts of any sort or even upon the manipulation of the extremities. The slighter movements have been called athetoid by some, but they present nothing in common with the true athetotic, vermicular movements so common in the cerebral palsies of children.

Fraenkel has recently emphasized the fact that *myoidism* is easily provoked, in the muscles which show diminished reflexes, by sharply pinching them, or by a smart light blow with a ruler, at right angles to their length.

Palsies.—The paralytic features in tabes are of two orders, and are to be clearly distinguished from the loss of coördinate power which constitutes the major motor difficulty. (1) One is due to the wasting of the muscles, a result of the involvement of the anterior horns of the cord, usually appearing late in the disease; (2) the second group, to which attention is here confined, is the result of organic or vascular changes in the cerebrum or cord. They appear in about one-fifth of the cases, and embrace hemiplegia, facial paralysis, lingual paresis, monoplegias, laryngeal palsy, and paraplegias, the last being due to focal disturbance in the cord. In addition, paralytic drooping of the lid constituting ptosis, one of the early manifestations of tabes, may be mentioned, but will be more fully described under disturbances of the ocular apparatus. The facial palsy also may be neuritic.

A peculiarity of these palsies, as pointed out by Fournier, who has tabulated them, is their usual benign and fleeting character. They may last some days or a few weeks, when they may completely and spontaneously disappear, but a minority of the palsies remain permanently. Some of these transitory cases undoubtedly are to be referred to hysteria, which not infrequently is a complication of tabes. The permanent cases are probably due to vascular or inflammatory accidents in the brain or cord; in other instances a neuritis may determine a localized loss of power.

Sensory Disturbances.—The sensory disturbances in locomotor ataxia are among the very earliest to appear, and persist in some form or other throughout the course of the disease. They are rarely the same in any two patients, and may vary almost infinitely in a given case.

Subjective Sensory Disturbances.—The first group of sensory disturbances are those of a subjective nature, regarding which we have to depend entirely upon the descriptions of the patient. They may be subdivided into those which are intermittent and those which are permanent. First and most important of the intermittent variety are the so-called *lightning pains*, which are experienced in the face, extremities, or trunk, but most commonly in the legs. They are described in the most vigorous language and the most striking terms by these unfortunate sufferers, are clearly atrocious in character, and are not associated usually with any evidence of disturbance in the part where they locate. Pains of a similar character, but somewhat less in intensity, and transitory like the lightning pains, are *lancinating, boring, burning, twisting sensations* of a painful character. In each patient an attack of such pain is likely to be followed by a similar attack in a similar location, and these attacks or crises occur in some cases with a degree of regularity every week, month, or year. Ordinarily, they are attributed to rheumatism, and it is not uncommon for patients to be treated for rheumatic disturbance for months and even years before the tabetic nature of the painful affection is recognized. A careful examination in a case marked by repeated painful attacks will almost invariably determine other evidences of tabes. The diagnosis should be made. When commencing early in the disease, the pains may disappear after the ataxia has become prominent, but if they do not appear in the early stage they are not likely to appear later. In some cases they persist through the entire duration of the disease.

Another variety of subjective pains are associated with the viscera, and constitute *anal, testicular, ovarian, urethral, vesical, gastric, laryngeal,* and *intestinal crises*, which will be taken up in the consideration of the visceral disturbances of the disease.

Tabetics also frequently complain of attacks of *muscular cramps*, which, like the lightning pains, have a tendency to come on in gusts, and frequently compel the patient to take to his bed. Their common location is in the muscular masses of the lower extremities or in the small of the back.

The *second class* of pains is more permanent in character, lasting weeks, months, or even years, perhaps appearing and disappearing several times during the course of the disease. One of the most important of these is the *girdle sensation*. The patient describes it as the feeling of a tight belt, and may frequently attempt to relieve it by loosening his clothing. It may be situated in the lower portion of the abdomen, or at the level of the umbilicus, or about the chest. It is usually rather narrow in its vertical extent, but may be of considerable width, giving rise to sensations of an iron or rigid corset. An analogous sensation is sometimes felt in the extremities as of a bracelet, or as if the legs or arms were wound with rope. Paresthetic sensations of a more or less persistent character are described by the patient as the creeping of ants or insects, a feeling of fullness in certain parts of the body, especially along the ulnar border of the forearm and hand, and in the lower extremities below the knees. In other cases the sensation is that of a cobweb or some light fabric upon the skin.

Sensory Disturbances Partially Objective.—*Analgesia* is one of the commonest manifestations of tabes, affecting not only the cutaneous extent, but the muscles, bones, and joints. Even dislocations and fractures, perforating ulcers, and other diseases of the deeper parts, as well as of the skin, are frequently unattended by pain. Very often the patient is unaware of the analgesia and is surprised to observe that a needle can be thrust deeply into the tissues without giving rise to any discomfort. This loss of the sensation of pain is frequently distributed in plaques upon the trunk and extremities without reference to the ordinary manifestations of the disease, and does not spare the surface of the head. It has a tendency to symmetry, affecting both upper extremities, both lower extremities, or the trunk bilaterally. On the head, however, there is a tendency to a unilateral distribution of the analgesia. On the trunk the most common location for the analgesia is over both pectoral regions, about the umbilicus, in each inguinal region, and over the shoulders. Frequently these analgesic plaques are marked by a hyper-

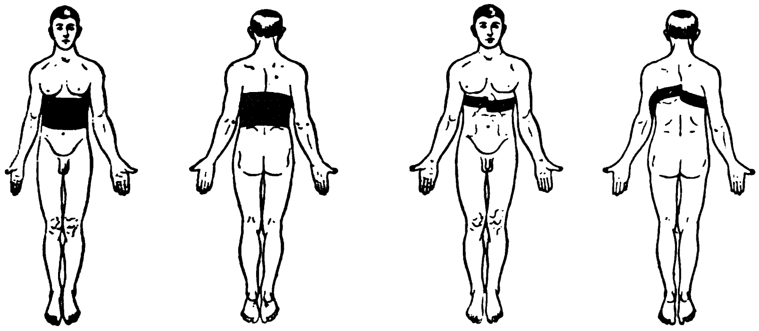
Fig. 187.—Tabetic cuirass of blunted tactile sensation.

esthetic border. On the upper extremities the analgesia most commonly affects the fingers or the ulnar border of the forearm. In the lower extremities it is the sole of the foot, the heel, and the toes; on the thighs the inner surface, corresponding to the adductors. The nerve-trunks, as the ulnar at the elbow and the external popliteal at the head of the fibula, are frequently found insensitive in very early stages of the disease. The testicle may also lose its normal sensitiveness to pressure.

On the trunk is frequently found what may be denominated the *tabetic cuirass*. In about four-fifths of the patients, even at an early stage, there appears a band about the chest, which may be most marked on the anterior or more commonly the posterior surface, or rarely confined to the pectoral regions. This area presents a diminished sensation to *touch* (Laehr, Patrick, Bonar). Its outline above and below is sometimes hyperesthetic. It varies in vertical width from three or four inches to an area which would be covered by a corset, and is sometimes associated with a girdling sensation. This cuirass distribution is not ordinarily one of analgesia, but of tactile loss, though it may be both. Its limits are those of the cutaneous extent of the spinal segments, and do not conform to the course of the intercostal nerves. These areas of tactile anesthesia have four principal localizations: (1) In horizontal patches or girdles on the trunk; (2) the internal surface of arms and forearms and ulnar borders of hands; (3) the perineal and genital regions; (4) the outer margins of the feet, outer sides of legs, and the anterointernal surfaces of the thighs. They are often related [1] to the painful disturbances of neighboring viscera.

Hyperalgesia is a common condition in tabes. It may appear in plaques similar to those of analgesia, but has less tendency to symmetry of distribution and is less persistent. These hyperalgesic plaques frequently are the foci of lightning pains and often appear during the painful crises. Hyperalgesia may be found not alone for the painful stimulus of the needle, but for cold and other sources of pain. Many patients in the early years of the disease find hot water intolerable in the bath and frictions by hand or towel almost unbearable.

In some instances the *stereognostic sense* is materially impaired and in a ratio disproportionate to other sensory modifications.[2] Thus, in the hands, which may be the seat of only slight paresthesia, a match-box may not be told from a coin and other familiar objects similarly mistaken or unrecognized.

Modifications of the cutaneous sensations are quite frequent. Commonly the *transmission of sensation* from the extremities is *retarded* so that the patient, when instructed to do so, does not indicate the perception of the pin-prick upon his shin or foot within a period of three, five, or even ten seconds, or more. In a general way the retardation of the transmission of sensation increases with the distance of the part from the head, not only because of the distance, but from the fact that the extremities usually present the greater disturbance of sensation, probably owing to the changes in the distal ramifications of the sensory nerves. A peculiarity is that the retardation of sensation may be dissociated. Painful sensation may be retarded, while that for touch is not, so that the patient feels the prick of a pin immediately as a touch and subsequently as a pain. Frequently patients are unable to distinguisn the character of the stimulating impression, recognizing a prick as a pinch. This, in other words, is an expression of the diminution of their sensitiveness. Tabetics may fail to properly locate the stimulus,—a pinch on the foot may be referred to the knee or to the opposite foot. In general, the tactile sensations are abolished later and to a less degree than sensations of pain, but they are also frequently modified, and this gives rise to additional difficulty in locomotion. The patients express themselves as having a feeling of walking upon a thick carpet, upon cushions, upon rubber, or other yielding substances.

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[1] Marinesco, "Sem. Méd.," Oct. 13, 1897.

[2] Rennie, "Br. Med. Jour.," Feb. 7, 1903.

It may be found that a stimulus not at first recognized is apprehended upon being repeated a few times with some rapidity, the *summation* of effects being competent to reach a sensorium cut off from a single impulse. Again, a stimulus at first competent may, upon repetition, fail to rouse the sensorium by *exhaustion* apparently of the conduction apparatus which, after a short interval of rest, again responds to the original excitation. Even the syringomyelic *dissociation of cutaneous* sensibility has been encountered in tabes, but commonly it is devoid of those exact and equal boundaries for all forms of sense anomalies which are found in the true syringomyelic syndrome.[1]

In all cases it is necessary to use the utmost caution in making tests of sensation, as already indicated in Part I. Some allowance also must be made for the intelligence and temperament of the given patient.

Disturbance of the Reflexes.—The *knee-jerks* are lessened, unequal, or more frequently abolished, and that at an early stage of tabes, in at least nineteen out of twenty cases. The lost knee-reflex, often called *Westphal's sign*, must be sought with great care, but in no instance should the patellar reflex be considered extinct unless the plan of reinforcement and all precautions are taken to elicit it. It may also be well to recall that it is diminished in advanced age, in sleep, by fatigue, in exhausting illness, and by any condition, such as a peripheral neuritis, that destroys the afferent and efferent paths or the spinal center. It is also possible that very rarely a healthy adult may be found without a knee-jerk. The *Achilles reflex* ordinarily fails with the knee-jerk. In fact, it may disappear before the knee-jerk is lost and constitutes a valuable early test. The reflexes in the upper extremity fail when the cervical cord is involved, and Fraenkel claims that the triceps reaction is lost as commonly and as early as the knee-jerk.[2] The *superficial reflexes*, such as the plantar, abdominal, dorsal, and scapular, are variable. The *iris reflex to light* is usually abolished early, but will be considered later with the disturbances of certain other organic reflexes, including those of the cremasters.

All muscles in the involved areas present a peculiar *lack of tonicity*, of which the reduced or lost tendon reflexes are a manifestation. This hypotone is readily demonstrated by the ease with which over-extension can be imparted to knees, ankles, and elbows, and in the great range of flexion at the hip and of abduction of the thighs.

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[1] Raymond, "Leçons," Paris, 1901.

[2] "Deutsch. Zeit. f. Nervenh.," July, 1900.

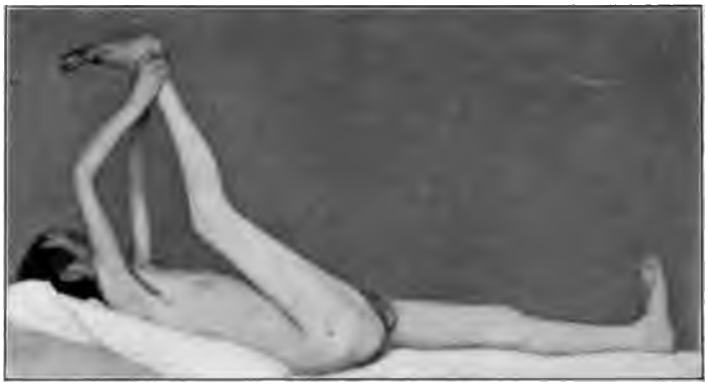

Fig. 188.—Abnormal range of flexion of hip-joint due to hypotonus in tabes.

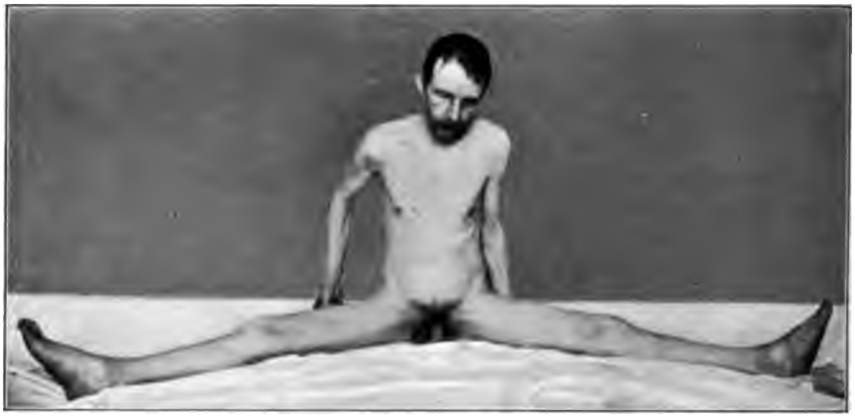

Fig. 189.—Abnormal abduction of thighs, the "split" position, due to hypotonus in tabes.

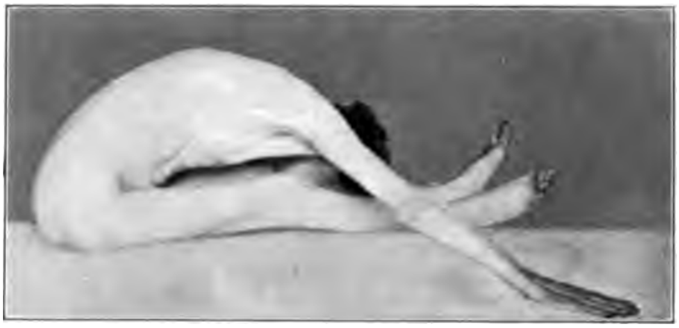

Fig. 190.—Abnormal flexibility of spine and hips in tabes due to hypotonus.

Disturbance of the Visual Apparatus.—Both the external and internal portions of the ocular mechanism are frequently impaired in tabes.

Ptosis and squints, usually unilateral, sometimes bilateral, are of common occurrence in the preataxic as well as in the later stages of locomotor ataxia. They may be and often are temporary and fleeting, almost momentary, but show a marked tendency to recur and occasionally are permanent. Careful questioning will commonly recall to a tabetic's mind some such ocular experience. Its temporary character is the best evidence of its tabetic, we may even say of its syphilitic, nature. Any of the extrinsic muscles of the eye may be selected by the disease, but those under the control of the third cranial nerve show more than their due proportion of paralytic disturbances. They may be gradually invaded, and a progressive external ophthalmoplegia results with permanent disability. *Lacrimation, exophthalmos, enophthalmos, nystagmus,* and reduced ocular *tension* on one or both sides has been noticed in rare instances.

The pupils are affected in the great majority of tabetic cases, and furnish some of the earliest and most important diagnostic symptoms. Every possible pupillary modification may be encountered in tabes,—*inequality, irregularity, miosis, mydriasis, sluggishness, loss of light reflex, loss of accommodation reflex, loss of reflex to pain,* and *absolute iridoplegia.* There is only one other disease that has a parallel in this matter of pupillary disturbance, and that is paretic dementia. The analogy, if not identity, of these diseases has been sufficiently indicated. These various pupillary disorders may be combined in any and every way. Sluggishness of the pupils to light and slight inequalities and irregularities of outline are usually encountered very early in the disease. Later, contracted pupils still responding to accommodative efforts but not to light, constituting the *Robertson pupillary sign*, are noted, and still later

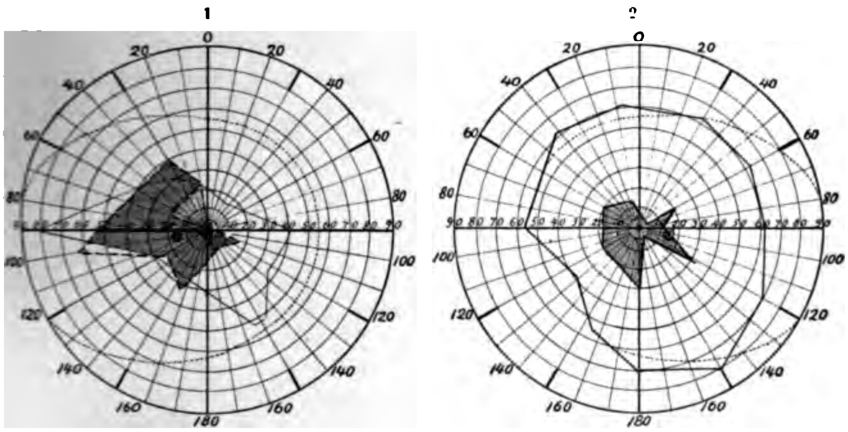

Fig. 191.—Contracted irregular visual fields in tabetic optic atrophy. 1, Left eye; unbroken line bounds form field, broken line ---- bounds field for red in which there is a blind scotoma; small central field is for green; 2, right eye; unbroken line bounds form field; small inner field is for green.

loss of reflex to pain, and eventually complete iridoplegia, with or without paralysis of accommodation, is often found. Dilated pupils may be subsequently contracted, but pupils once contracted to an extreme degree very rarely again dilate widely. The fixed pupils of tabes resist agents that ordinarily control the pupillary muscles, and if forced from their abnormal proportions, as by belladonna, return to them only after several weeks. The dissociation of the light and accommodative reflex is, perhaps, the most important of all ocular symptoms, and appears early in over one-half of all cases. Over seventy per cent. of tabetics show some pupillary disturbance.

The optic nerve degenerates in about ten per cent. of tabetics, and this occurs in those patients who have shown ocular palsies more frequently than in others. The optic atrophy is usually bilateral, but has a tendency to attack the left eye first. Its natural termination is in blindness. In rare instances it comes to a standstill, or even recedes a trifle. It may cause blindness in a few months, or many years may be required for the extinction of sight. As a matter of fact, the optic atrophy may have advanced to a considerable development before the patient notices any visual impairment. The order of symptoms is usually: (1) A contraction of the color-fields, green, red, and blue fading in the order named; (2) the form-field shrinks, and (3) vision begins to diminish. The retraction of the field is usually most pronounced on the temporal side, but may be irregularly concentric. In occasional instances hemianopsia or quadrant defects have been observed, and even central scotomata. These partial fields are, perhaps, properly attributed to a retrobulbar neuritis. The characteristic ophthalmoscopic picture from the first shows a blanching of the papilla, which becomes grayish or bluish-white and pearly. Its border is sharply defined, and sometimes it is stippled by the cribriform markings. Eventually the vessels diminish in size, first the arteries, then the veins.

Optic atrophy may be a very early tabetic feature, and usually appears either before or during the early portion of the ataxic stage, while the patient is still walking fairly well. It is a clinical fact of much value that, as a rule, the cases developing optic atrophy early do not show much ataxia, and the locomotor difficulties are only those of blindness. When appearing in the ataxic stage there is little or no further increase of incoördination, and in certain cases the ataxia almost or quite disappears. Benedikt says it invariably subsides.

Auditory Symptoms.—Morpurgo[1] has shown that eighty per cent. of tabetics present auditory defects which may appear early or late in the disease.

The auditory nerve is subject to a degeneration similar to that noted in the optic and about as frequently. In other instances the disturbance is in the middle or external ear. The *impairment of hearing* is frequently insidious and extremely slow in reaching a complete degree, but in some cases comes on with rapidity or even abruptly. It is usually bilateral but commonly more marked on one side than upon the other. The distinction between disease of the nerve and disease of the conduction apparatus is determined by Rinne's test (see page 64). Weber's test, which consists of placing the handle of a vibrating tuning-fork on the vertex, enables the patient to thus hear the note better in cases of middle or external ear disease than when the nerve is affected. The disease of the middle ear is, perhaps, sometimes of a dystrophic sort.

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[1] "Archiv. f. Ohrenheilk.," 1890.

Many tabetics are troubled with *tinnitus* and vague subjective auditory sounds. Others have constant or paroxysmal attacks of *aural vertigo* that may be sufficiently severe to provoke vomiting and great prostration. Auditory *hyperexcitability* to electric currents was found by Marina [1] in eight out of eleven cases of tabes, which also indicates the frequency of disturbance of the auditory apparatus in locomotor ataxia. Bonnier [2] attributes to labyrinthine disturbance many of the common signs and symptoms of the disease. He enumerates deafness, vertigo, ataxia, nystagmus, diverse disturbances of oculomotricity, pupillary changes, and a large number of others, and found labyrinthine irregularity in 80 per cent. of cases examined.

The senses of *smell* and of *taste* have also been found impaired in rare cases, even to the point of complete loss. A careful examination for similar defects in all cases would probably show them to be somewhat common. Klippel and Julian [3] have reported nasal crises marked by curious sensations in the nose and nasopharynx and violent bouts of sneezing.

Visceral Disorders.—The visceral features of tabes are among the most interesting and constant manifestations of the disease, and among those most frequently overlooked and misinterpreted by the physician. They consist, for the most part, of paroxysmal attacks attended by pain and disturbed function of some viscus, as the stomach, intestine, or bladder, and are called *tabetic crises*. Other visceral disorders are of a continuous character, and are attributable to impairment of the nervous and vasomotor control of the parts. Though any viscus may be affected in tabes, the crisis features for the given patient are usually limited and uniform in their manifestations.

The Stomach.—*Gastric crises* in tabes are very common. They are characterized essentially by pains and vomiting. The *pains* are located in the pit of the stomach and often radiate in various directions. They are occasionally referred to the heart; indeed, angina pectoris may appear with them. They are intense, and sometimes so severe as to seem unbearable, and may actually cause insensibility. Sometimes they strike through to the back or flash into the flanks and through the abdomen. The *vomiting* is repeated and intractable. Sometimes it is attended with excessive straining, and again the gastric contents are ejected with very slight eructative efforts. The *vomitus* at first consists of undigested food and then of gastric mucus in large amounts, and finally of bilious mucus in the protracted attacks. The vomiting is frequently repeated, only a small amount being ejected at a time after the first efforts. The slightest ingestion of food or liquid of any sort promptly provokes a repetition of the emesis. Sahli, Hoffmann, and others have shown that there is hyperacidity due to an increase of hydrochloric and lactic acids, most marked at the beginning of the attack and gradually diminishing as it proceeds.

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[1] "Archiv. f. Psych.," t. xxi, p. 156.

[2] "Nouv. Icon. de la Salpêt.," 1899.

[3] "Rev. de Med.," Jul. 10, 1900.

The gastric crisis is usually attended by a state of marked *prostration* that may even recall the collapse of the algid stage of cholera and is usually equal to that of severe seasickness. The patient, cold, blanched, and covered with profuse perspiration, presents the appearance of severe shock.

Gastric crises, like all the critical manifestations of tabes, are of *sudden onset* and *abrupt termination*. They may last an hour or two, or several days or weeks without intermission. In spite of the great thirst that attends prolonged attacks, the smallest amount of liquid is not tolerated by the stomach, and alimentation *per os* is out of the question. Suddenly the patient may feel hungry, the pains may abruptly cease, and both food and drink may be taken freely without further disturbance.

Gastric attacks often occur in the preataxic stage of posterior sclerosis and are attributed to all sorts of indigestion, but it is difficult to identify any actually determining cause. They sometimes occur but once in a given case. Ordinarily, they are repeated, and sometimes with regularity, every few weeks or months, or even daily. After several years they may diminish in frequency and definitely cease, or they may persist throughout the entire course of the disease. They may even cause a fatal termination.

Variations are not uncommonly encountered in which the gastric crisis may be unattended by much pain or the pain may be excessive, and vomiting slight or absent. They may closely simulate hepatic or nephritic colic, or be marked most by the generation of an extreme flatulency. Occasionally there is considerable blood in the vomitus, giving rise to the suspicion of gastric ulcer.

The intestine is often disturbed in tabes. In some cases there is persistent causeless *diarrhea*, marked by frequent slight liquid stools, not attended by colics or pain. In other instances *constipation* is beyond control and fecal accumulations, apparently even above the colon, cause distressing and persistent complaint. Intestinal or *rectal tenesmus* occasionally drives the patient almost distracted. There is a constant desire to defecate, but efforts are abortive, or only result in a small passage. The repeated imperative character of these attacks, with their apparent causelessness, should serve to distinguish them. A rectal examination is usually negative. Rectal crises may simulate dysentery, particularly as considerable blood may appear in the stools. In many late cases the *anal sphincter* is incompetent to retain fluid bowel-contents or injections.

The urinary apparatus presents some of the earliest indications of tabes. Among the first symptoms is a *difficulty in starting* the urinary discharge and in *completely evacuating* the bladder. This condition must be intelligently investigated, as it may escape the patient's attention or be deemed of no significance. As a rule, tabetics have to make forceful abdominal expulsive efforts to complete micturition, and in some cases this is aided by pressing the hands deeply into the lower belly-wall. Not infrequently after protracted waiting, the feeble stream suddenly ceases to flow, or after the act is thought to be completed a small amount of urine wets the clothing. Complete *retention* is rarely encountered, but daily catheterization may be required in some cases. Similarly partial or complete *incontinence* is met with, but what is more common is an inability to control the flow of a few drops or more of urine if the slightest desire to urinate arises, or even the thought of it occur. Very frequent urination may be due to a weak *sphincter* or to *cystitis*, which often arises from retention.

The *character of the urine* in tabes is frequently altered. *Glycosuria* is somewhat common among tabetics. The hereditary relation of tabes and diabetes has been already suggested. The medullary lesions of tabes may stand for something in this relation. In some cases there is a *quantitative diminution* of the urea, or phosphates, or chlorids. Sometimes the quantity of urine is notably lessened, sometimes greatly increased in a paroxysmal manner.

The mucous lining of the urethra and the bladder may be insensitive, but that does not prevent their being the seat of atrocious painful attacks that constitute *vesical crises*, or, extending to the lumbar regions, suggest the term *nephritic crises*. During such attacks the patient is constantly tormented with the desire to urinate, but fails to express more than a drop or two at a time. Meantime the colicky, darting, intense pains about the neck of the bladder and down the urethra or thighs may give rise to intense suffering. Lightning pains in this region are not infrequent and vesical and rectal crises are often associated.

The generative functions frequently undergo modifications in tabes. In over one-half the cases there is a *loss of sexual appetite* and more or less *impotence*. Erections either completely default or are partial, and ejaculation does not take place. *Impotence* may develop very early, and sometimes is the first symptom to attract the patient's notice. In a majority of these cases there is an *early genital excitement* that has sometimes led to sexual excesses and has perhaps contributed to the idea of the causal rôle of such practices. The same thing is seen in general paresis. Other reflexes than the genital may be exaggerated in the initial period of tabes, as witness the increased knee-jerk that appears in very exceptional instances, and the spasmodic action of the bowels, rectum, and bladder.

With the loss of generative aptitudes in tabes we usually find a diminution or complete extinction of the *cremasteric reflex*. As there is a loss of appetite these patients make no complaints, contrasting sharply with the sexual neurasthenic, whose cremaster also is likely to be extremely active. In the same tabetic condition the *bulbocavernous reflex* of Onanoff, or the *virile reflex*, as it is called by Hughes, is usually, if not always, absent. To secure this reflex the index-finger of the examiner is firmly placed over the bulbar portion of the urethra at the angle of the scrotum and perineum, and the mucous membrane of the corona glandis lightly pinched. The palpating finger will distinguish the contractions of the bulbocavernous and ischiocavernous muscles. This reflex is said to be invariably present in healthy males, and even in other nervous diseases, whenever complete erection is possible.

Testicular analgesia has been already mentioned. It is present in about four-fifths of all tabetics, and is not infrequently attended by *atrophy of the testicle*.

In female tabetics there are analogous changes in the sexual sphere. In both sexes the genital organs are occasionally the site of painful crises that are commonly misunderstood, especially as they are prone to occur in the preataxic stage of the disease.

The Respiratory Apparatus.—*Hyperesthesia* and *anesthesia* of the soft palate and diminution or increase of pharyngeal and laryngeal sensibility may be found separately or variously combined in cases of locomotor ataxia. Oppenheim has described *pharyngeal crises*, consisting of rapidly repeated, noisy, and very painful swallowing efforts that are involuntary. They last from a few minutes to a half-hour and are attended by some facial cyanosis and abundant perspiration. The writer has observed them in a case with progressive involvement of the cranial nuclei.

Laryngeal crises are tolerably common in tabes and vary greatly in degree and intensity in different cases. Sometimes there is a noisy, croupy inspiration, to which a cough may be added that strongly suggests whooping-cough. There is more or less dyspnea, pain, anxiety, and depression. In some instances the dyspnea seems to be absolute and the patient falls suddenly, cyanotic, unconscious, and convulsed. After a few moments the laryngeal spasm yields and full consciousness immediately returns. This form of laryngeal crisis has been called the *laryngeal stroke* by Charcot.

In patients subject to these crises they may be provoked by slight irritation of the laryngeal mucous membrane by mechanical or other means, and in some instances by pressure of a sensitive point on the side of the neck between the lower border of the larynx and the sterno-mastoid. Like other tabetic crises, they may increase in severity and frequency or grow less intense as time passes, and they have the same tendency to recur. Though giving rise to alarming symptoms, they are, ordinarily, without danger in themselves. They appear to be due to unusual sensitiveness of the laryngeal surfaces.

Laryngeal palsies are encountered in tabes and may or may not be attended by laryngeal crises. The palsy may affect any of the laryngeal groups of muscles on one or both sides, but seems to exercise a preference for the dilators. Corresponding respiratory and vocal symptoms follow. The nerves, roots, and bulbar centers have been found variously degenerated, and the muscles themselves secondarily atrophied. *Bronchial attacks*, marked by spasmodic cough and respiratory difficulty, are rarely encountered.

The Vascular Apparatus.—Taking into view the fact that syphilis is one of the commonest causes of vascular deformities and disease, the frequency of arterial sclerosis and cardiac abnormalities in locomotor ataxia is not surprising. Aside from the cerebral vascular accidents arising from this source, angina pectoris, associated sometimes with gastric crises, is encountered. *Valvular disease*, affecting both the mitral apparatus, usually in the form of insufficiency, and the aortic valve, mainly by stenosis, is found with considerable frequency. In 300 cases

Limbach[1] found mitral insufficiency alone in 2 cases; with aortic stenosis once; 2 cases of aortic insufficiency were noted, 1 of aortic insufficiency and stenosis, and 1 aneurysm of the aorta,—7 in all, and all syphilitic. Lesser[2] found aneurysm in 19 out of 96 cases, 20 per cent. A rapid pulse, from 100 to 120, is not uncommon. The *blood* may be normal or impoverished and frequently contains cholin.

The temperature in tabes is normal or only shows variations dependent upon intercurrent associated or secondary disorders. Pel[3] has reported a case with crises of high temperature and rapid pulse lasting about twenty-four hours. There were also coryza, lacrymation, photophobia, and lancinating pains in the face and eyes. Oppler[4] reports a similar case.

Trophic Disorders.—The disturbance of nutrition in tabes finds some manifestation in nearly every case, and there is no tissue or structure that may not be affected. The great majority of tabetics, and perhaps all in the later stages, show a depravity of the *general nutrition* that can not be explained by their physical inactivity, by pains, or by a syphilitic cachexia. In the preataxic and ataxic stages the general malnutrition is frequently well marked, but exceptionally the tabetic is plump, ruddy, and apparently vigorous.

Osseous System.—On the part of the skeleton tabetic dystrophy presents two striking clinical manifestations,—*spontaneous fractures* and *dystrophic arthropathies.*

Spontaneous fractures in tabes are more frequent than might be supposed, as they are not by any means always referred to their proper origin. It would appear that they are more common among women than in men. They may occur in the very early stages of posterior sclerosis, during the ataxic stage, or in the last phase of the disease. Their most frequent site is the femur, especially the shaft; the leg bones, and those of the forearm; but any long bone may present this accident, and even the vertebral bodies are sometimes thus affected. The fractures may be repeated or multiple in the same subject. Separations of epiphyses and of the bony insertions of muscles are also encountered.

Tabetic fractures are marked by the practical *absence of pain* in the affected part, and by the usually *insignificant force* that occasions them. They have followed merely the crossing of the knees or have taken place while the patients have been calmly walking on a smooth surface. Union takes place readily, but, owing to the lack of pain, which ordinarily reinforces the immobilization of the parts by splints, movements of the limb are not inhibited. *Shortening* and *extensive callus* are the natural results.

The bones in tabes and paretic dementia present a certain *fragility* which lays them liable to fracture during life. To the naked eye they often present a *porosity* and a shrinking of the compact substance, with an *increased sponginess* of the more open structures and sometimes an enlargement of the medullary canal. Microscopically, the Haversian canals are dilated and some *decalcification* is evident. The *osteoblasts* are shrunken and sometimes show fatty degeneration, while the medullary substance is increased to an amount corresponding to the diminished osteal portion. This results in an inversion of the ordinary ratio between the organic and inorganic elements. Normal bones are about two-thirds inorganic substance; tabetic bones are about *two-thirds organic matter*. In other words, these bones present a *rarefying osteitis*. By some this is attributed to changes in the nutrient artery and nerve, which have been found sclerotic and neuritic. For others the lesion is a manifestation of the trophic disturbance arising from modification of trophic cells in the spinal ganglia and cord.

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[1] "Deutsch. Zeit. f. Nervenheilk.," 1895.

[2] "Berlin. klin. Woch.," Jan. 25, 1904.

[3] "Berlin. med. Woch.," Jun. 26, 1899.

[4] "Berlin. klin. Woch.," 1902.

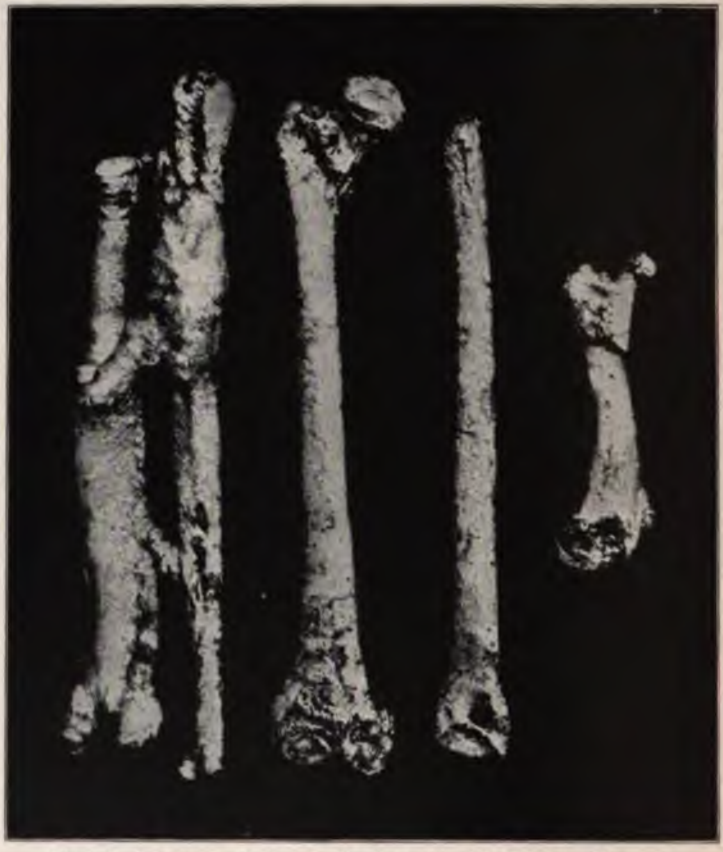

Fig. 192.—Spontaneous fractures and arthropathic disintegrations (Charcot).

Tabetic Arthropathy.—One of the early features of tabes is an *abnormal range of joint-motion*. This may be observed even in the pre-ataxic stage, but is usually developed after incoördination has appeared. Putnam[1] was among the first to call attention to the fact that the joints in tabetics could be forced into extreme flexion or extension without producing much or any pain, and attributed the fact to analgesia. Fränkel and Faure,[2] in a fuller study of the matter, show the extreme range of motion that is customarily found in the joints in this disease.

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[1] "Bost. M. and S. Jour.," Aug. 29, 1895.

[2] "Nouv. Icon. de la Salpêt.," July, 1896.

They found that in locomotor ataxics the foot, wrist, elbows, and fingers are similarly affected. Their patients could execute at the first attempt the "split" attitude that acrobats only attain by years of practice. They attribute this abnormal flexibility to muscular and joint analgesia in part, but principally to a loss of the muscular tone. This condition of the joints places them at a certain disadvantage, so that the joint-surfaces are not properly coapted and ligamentous stretching is often induced. Taken with the incoördination of movement, there is little doubt that the joints are subjected to unusual traumatic twistings and shocks. In addition, there is present the trophic disturbance marked by fragility of bones, and out of it all arises the *tabetic* or *Charcot's joint.* Its frequency in syringomyelia adds force to the belief that the spinal lesion is the principal cause, the traumatism the excitant.

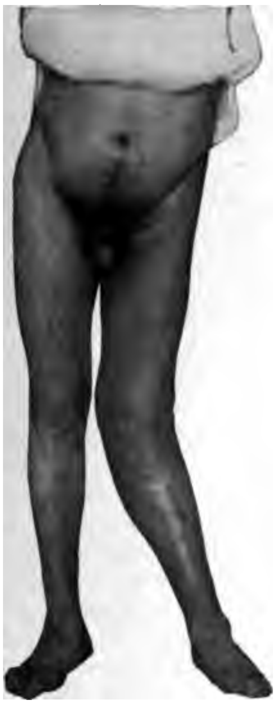

Fig. 193.—Tabetic arthropathy of the left knee.

A *tabetic arthropathy* is marked at first by (1) *rapid* or even *sudden onset*, (2) entire or nearly entire *absence of pain* and tenderness, and (3) *enormous swelling* of the adjoining parts. The patient, while walking or using a member, may notice a sharp cracking in a joint and find that the limb feels heavy and more unmanageable than usual. Shortly—that is, in the course of a few days—not only the joint, but the entire segment of the limb is greatly swollen. This swelling may be the first thing to attract attention. The parts are found tense but cool, and devoid of redness and tenderness. The swelling is extreme, but not boggy, and does not pit on pressure. Movements of the joint and its employment in no way inconvenience the patient. In *favorable cases*, after a little time, the swelling subsides and all trouble disappears except a little thickening about the joint and some creaking in the articulation. There is, however, a great tendency to the *recurrence* of aggravated and more lasting attacks. In *severe cases* the swelling does not disappear so promptly, but becomes circumscribed about the joint in a more or less *globular* form, and the joint-surfaces as well as the ligamentous structures undergo *disintegration*. Finally, the limb-segments may be united only by soft tissues that permit painless motion and circumduction in every direction. Added to the joint-changes, we may have spontaneous fractures, epiphyseal separations, suppuration, and even the protrusion of the bones through the skin. Old tabetic joints present merely a bag of bone-fragments where articulations were formerly located.

In 132 cases of tabetic joint disease Kredel found arthropathies occurring 21 times in the prodromic or preataxic stage, 38 times between the first and fifth year of the disease, 32 times from the fifth to the tenth year, and 41 times after the tenth year. They occur in 3 or 4 per cent. of all cases, and more frequently in women than in men.

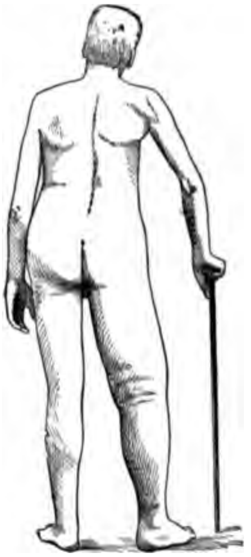
Fig. 194.—Tabetic arthropathy of right knee, early stage, with edema of the entire extremity (Souques and J. B. Charcot).

The *localization* of joint-disease in tabes is mainly in the large joints, but no articulation is exempt. Flatow collected 139 cases, in 41 of which there were bilateral arthropathies. The order of frequency was as follows: knee, 60; foot, 39; hip, 38; shoulder, 27; elbow, hand, fingers, and maxilla, 4 to 6 times each.

Upon section of these joints the *capsules* are found dilated, often ruptured, and in old cases completely destroyed. The *ligaments*, especially those within the joints, as at the knee and hip, are diseased or have disappeared. The *synovial membrane* is thick, rough, and often adherent to the surrounding parts; later it may be absent. It may contain bony particles and osseous nodules. The *joint-fluid* is thin and clear or yellowish and exceptionally purulent or bloody. At first it is abundant and infiltrates the parts about the joint, into which it escapes through the ruptured capsule and accounts in some cases for the great swelling in the limb. It may contain floating bodies in large number, bony particles, and detritus.

The ends of the *bones* and joint-surfaces may be either (1) eroded, as is most usual, and greatly reduced in all their dimensions, even to the complete destruction of several inches of their length, or (2) may present the hypertrophic exaggeration of an arthritis deformans. These two types may be combined in the same joint. The rule is that the hypertrophic form occurs in the knee, the atrophic variety at the hip and shoulder. The disintegration of the joint may be increased by intracapsular fractures or by fragmentation of the eroded shafts and separated epiphyses.

Juergens has found that in most tabetics nearly *all the joints* show capsular enlargement, elongation of ligaments, vascular dilatation, and some synovial roughening. The preponderance of such joint-disease in the lower extremities depends, perhaps, upon their more exacting and vigorous use, and consequent liability to strains and traumatism and upon the major lesions of the lumbar cord.

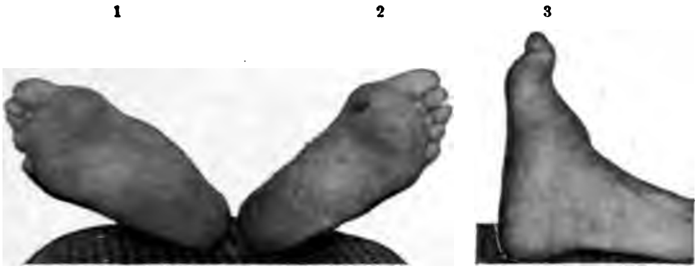

Fig. 195.—Tabetic feet. 1 and 3 show deformity due to arthropathic disintegration of the tarsus; 2 has a perforating ulcer under head of first metatarsal.

Trophic Cutaneous Disorders.—In tabes there are a number of *trophic dermatoses* that are of rare occurrence and insignificant importance. *Herpes zoster* is of more frequent appearance, and finds its favorite location on the trunk, rarely in the distribution of the trifacial. The *epidermis* of the extremities, especially the upper ones, is sometimes hypertrophic. *Hyperidrosis*, *anidrosis*, and the *loss of nails* or *teeth* have been occasionally noted.

Perforating ulcer is not an uncommon accident in tabes, and, though painless, is of considerable importance. It is usually situated in the foot, but may occur in the hand, and some authors have conceived that maxillary and even cardiac and visceral ulcerations were in some instances of the same character. It usually begins as a callus or corn on the sole of the foot, under the ball or under the base of the fifth metatarsal, or at the heel. Ulceration follows, and, if neglected, may denude bone and lead to exfoliation. The ulcer is indolent, persistent, and refractory to any treatment if pressure be not removed. Not uncommonly the toe-joints or those of the foot present dystrophic conditions at the same time. *Bedsores* only appear in terminal stages, and present nothing of a special nature. After an attack of lightning pains the part in which they are principally located may sometimes present a more or less distinct *ecchymosis*.

Muscular Atrophies.—In addition to the invariably diminished *muscular tone* that has already been mentioned, and the rare occurrence of *fracture of a tendon*, some tabetic cases present notable *amyotrophia*. This should be sharply distinguished from the emaciation, flaccidity, and incoördinate feebleness that are very common in advanced tabes. As a rule, the muscular masses and contours are well preserved until the ataxia is well developed, and often until the patient has for long been unable to walk, but in rare cases, perhaps in one per cent., localized muscular atrophy appears. Its *common seat* is in the lower extremities, especially invading the foot- and leg-muscles, and is usually bilateral. The upper extremities may be invaded, particularly the small muscles of the hands, or even the forearm, arm, and shoulder. The cranial nerves are sometimes similarly affected. The motor portion of the trifacial and the hypoglossus are the ones usually selected.

The *onset* of such amyotrophies is usually insidious. They present variously modified electrical reactions, the full reaction of degeneration being rare. Once established, the muscular atrophy of tabes remains fixed, and does not invade group after group of muscles, as do various progressive amyotrophies. The resulting *deformity* in the foot is due to pure, flaccid atony without contracture. The foot drops by its own weight and the pressure of bed-covering into an equinovarus. In the hands some clawing may be induced and the thenar eminence is likely to disappear. Hemiatrophy of the tongue follows hypoglossal involvement.

In a general way, according to Marie, tabetic muscular atrophies may be divided into two groups: (1) Those appearing at an advanced period of the disease, presenting a symmetrical distribution, rarely marked by fibrillary twitchings; (2) those occurring often in the earlier stages of the disease, usually unilateral in distribution, and marked by fibrillary contractures and sometimes by the reaction of degeneration. The first group embraces those atrophies confined to the distal portions of the lower or upper extremities, and recalls the conditions found in multiple neuritis. The second group contains lingual hemiatrophy, localized atrophies of the shoulder, of the back, of the hand, and one-sided involvement of cranial nerves. They are analogous to lesions of the nuclear gray matter. Both the central and peripheral lesions are found, and in the associations above indicated. The wasted muscles present the usual histological change, due to degeneration in the lower motor neuron.

Cerebral Disturbances.—In addition to the vascular cerebral accidents, with resultant palsies and the involvement of cranial nuclei, tabetics are subject to other cerebral disorders. These embrace the many possibilities of cerebral syphilis, and particularly paretic dementia. Apoplectiform and epileptiform attacks, or any unusual forgetfulness, exhilaration, expansiveness, or stupor should at once arouse suspicion of this fatal cerebral disorder. It should be studied in this connection, and is set forth in the second part of this work. On the whole, notable psychical disturbance is a rarity, but some degree of apathy, of indifference, is usually to be observed. This pertains particularly to themselves, their disease, their almost hopeless prospects. It is not much modified, even by the most atrocious suffering, and persists even in the stage of complete helplessness. Tabetics, however, often manifest large mental activities and retain their business capacity to the end.

Tabulation of Tabetic Symptoms.—The following table of tabetic symptomatology is that of Limbach,[1] based upon 400 cases selected from the private practice of Professor Erb.

In the first table the usual early symptoms are arranged in their order of frequency. There is an overlapping, as frequently two or more are alleged to have come on at or about the same time:

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[1] "Deutsch. Zeit. f. Nervenheilk.," 1895.

EARLY SYMPTOMS OF TABES.

	FIRST SYMPTOM.	SECOND SYMPTOM.	TOTAL.
Lancinating pains	283 times	65 times	348 times.
Cystic weakness	90 "	119 "	209 "
Feeling of weakness in legs	78 "	113 "	191 "
Paresthesia in legs	74 "	10 "	84 "
Girdle sensation	34 "	44 "	78 "

The relative frequency of various objective and subjective symptoms as observed in these 400 more or less complete histories is shown in the following table:

RELATIVE FREQUENCY OF TABETIC SYMPTOMS.

			PER CENT.
1.	(*a*) Failure of knee-jerk and Achilles jerk	92.0	96.25
	(*b*) Alteration in these reflexes	4.25	
2.	Swaying with eyes closed		88.75
3.	Lightning pains		88.25
4.	Disturbances of the bladder		80.5
5.	Ataxia of the lower extremities		74.75
6.	Changes in the pupillary reactions		70.25
7.	Paresthesia of lower extremities		64.5
8.	Feeling of weakness in the legs		62.25
9.	Diminution or disappearance of sexual desire		58.25
10.	Alterations in size of pupils		48.25
11.	Delayed conduction of pain		36.5
12.	Slight analgesia of lower extremities		33.75
13.	Girdle sensation		31.0
14.	Transitory double vision		26.5
15.	Diminution of sense of touch on lower extremities		23.25
16.	Paresthesia in ulnar distribution		16.5
17.	Ocular paralyses and ptosis		16.0
18.	Optic atrophy		6.75
19.	Persistence of painful impression in the legs		6.0
20.	Various crises		5.25
21.	Arthropathies		1.75

Course and Varieties.—Ordinarily speaking, the onset of tabes is extremely insidious and its course very slowly progressive. For purposes mainly of description it may be divided into the *preataxic*, the *ataxic*, and the *paralytic* stages. These indefinitely blend, and, as has been repeatedly indicated, many symptoms, commonly of the later periods, may appear precociously in the early phases of the malady. From the tabulation of symptoms, as well as from their individual description, it will have been noted that pains are among the earliest indications of tabes, and these may persist for years, even for a dozen years, before the prominence of other symptoms determines their character. Usually only when visceral crises, vesical weakness, ocular palsies, insecurity upon the legs, or inability to walk in the dark or down a stair or to stand securely while washing the face have seriously attracted the patient's attention is a properly directed investigation instituted. Then his "rheumatic pains," his "gouty pains," his "neuralgic attacks," his "bilious attacks," take their proper place. At that time a search of the cutaneous sensibility usually reveals its impairment in the feet and legs, the knee-jerks are absent, the pupils sluggish or inactive to light, and the ataxia can be demonstrated by the usual tests. In certain rare benign cases the disease never progresses beyond this point.

In the *second period* the ataxia increases and is apparent at a glance, but may be practically confined to the lower extremities for from two to six years or more. Then it may invade the upper extremities progressively. A host of sensory, motor, trophic, and visceral symptoms are present, varying in every case but usually consistent and uniform in the given instance. Even at this point the disease may halt in its progressive course. Usually the lack of motor control becomes greater and greater, walking more and more laborious, the ataxia intensified, and finally the patient is brought to the bed or chair in the *third period* of the disease. Now accentuation of intestinal and especially of vesical disturbance and the depreciation of the general physical state, taken with the helplessness, make the picture pitiful indeed. All its colors may be deepened by the atrocious pains that sometimes pursue the unhappy victim to the last. Cystitis looms as a constant menace to life, and any intercurrent affection is likely to be promptly fatal. From ten to twenty or thirty years may be consumed in the history of tabes or it may unroll its panorama of symptoms within two or three.

Tabes presents numerous variations from the wide symptom group that may be considered its common type. The *cervical form* presents pain and ataxia first in the upper extremities, which may also show trophic changes. Little static ataxia or incoördination of locomotion may be presented. The knee-jerk may even be retained, but that is rare. In the *bulbar form* we encounter early symptoms on the part of the cranial nerves, pharyngeal and laryngeal crises, optic atrophy, and ocular palsies. The tendency of ataxia to disappear upon the appearance of optic atrophy or for the disease to then become stationary furnishes a definite group of cases.

From another view-point, cases may be considered *benign* and *grave*. As has been indicated, the tabetic process may stop at almost any point. or after a lapse of years may again slowly advance. Some cases that are marked by intensely painful manifestations seem to be of slow evolution. This may be a way of saying that cases presenting a protracted first and second stage, to which the lightning pains and intense crises are usually confined, less rapidly disable the patient. On the other hand, cases of tabes are grave by the rapidity of their development and the intensity and generalization of their symptoms, due to the wide-spread underlying sclerotic process. Acute cases may confine the patient to bed in a few months. Leyden describes cases of extreme rapidity. Active syphilitic processes in brain and cord may be added and the patient at once overthrown. A marasmic state or the appearance of paretic dementia constitute conditions of extreme gravity.

Diagnosis.—The diagnosis of tabes in the full-blown ataxic stage rarely presents any considerable difficulty. Confusion usually arises by mistaking other diseases for tabes and in misinterpreting the early manifestations of tabes for those of slighter ailments. Gastric, laryngeal, intestinal, vesical, urethral, and all visceral crises, if present in the preataxic period, are almost invariably referred to the wrong source. Their repetition without clearly competent exciting cause or local lesions should always arouse suspicion of posterior sclerosis, which, if present, will not fail to present other symptoms and signs. The same is true of repeated attacks of severe pains of a lancinating or lightning-like character. The occurrence of these in a patient where syphilis is even suspected to have been present should direct attention to the spinal cord.

If the knee-jerks are gone or very unequal, or even greatly reduced, it should add to the suspicion of tabes. If, now, the Robertson pupil is detected, or even sluggishness of the pupil to light is clearly made out, the diagnosis may be considered established. The detection of several or many of the usual subjective and objective features of the disease will confirm it. Among these, too much importance can not be given to vesical disturbances and variations in the sexual sphere. The determination of lymphocytosis of the spinal fluid and cholin in the blood has considerable significance.

The condition most usually mistaken for tabes is *multiple neuritis.* The differential indications are tabulated on page 324. Unfortunately, a group of maladies of a similar, if not identical, character with multiple neuritis has been denominated *pseudotabes.* We thus encounter cases described as toxic pseudotabes or *neurotabes,* due to alcohol, arsenic, or other poison, diabetic pseudotabes, neurasthenic pseudotabes, and syphilitic pseudotabes. They often present the symptoms of multiple neuritis, with unusually severe root pains, ocular disturbances, or other symptoms that suggest tabes. The absence of well-marked crises, of the Robertson pupil, of sphincteric weakness, of pure incoördination without paresis, and the history of the onset of the disease, the uniformity and invariability of sensory symptoms, the history or presence of the toxic cause, and the usual early presence of some muscular wasting and the reaction of degeneration should distinguish pseudotabes from posterior sclerosis.

Paraplegias are marked usually by definite areas of dysesthesia, the reflexes are exaggerated, and clonus common, unless the cord is completely divided, when all motion below the lesion is abolished. This is not the case up to the last moment in tabes.

Cerebellar tumor may present some analogy at first sight, but usually we have choked optic discs, increased or merely reduced reflexes, intact sensibility, retracted head, and occipital pain.

Insular sclerosis presents some symptoms found in tabes, but is distinguished by the intention tremor, nystagmus, scanning speech, usually preserved and often increased knee-jerks. The sensory disturbance and painful manifestations are insignificant.

Syringomyelia usually affects the upper extremities first, and may be mistaken for cervical tabes. The dissociation of cutaneous sensibility is its chief characteristic, but even this has been found in tabes. Scoliosis, mutilations of the fingers, local atrophies occurring early in the disease, without incoördination and usually with increased knee-jerks, taken with a full history of the case and the careful delimitation of the sensory disturbances, should make the diagnosis exact.

Prognosis.—The diagnosis of tabes largely conveys the prognosis. When the degenerate changes that constitute the so-called sclerosis have taken place, restitution *ad integram* is, as far as now known, out of the range of possibilities. While, as a general rule, the disease is a steadily progressive one, there are many exceptions. In the enumeration of varieties of tabes attention was called to the benign cases and the possibility of the degenerative changes coming to a standstill at any period of development. This renders it the more difficult to estimate the value of medication. It has also been indicated that when optic atrophy appears the locomotor difficulties usually do not increase. The rate of advancement of the disease from its inception is some index of the rapidity of its future progress. Bulbar symptoms and the indications of developing paretic dementia at once render the outlook most gloomy. The duration of life is not, however, abridged as much as might be supposed. Marie and Moguot,[1] on a basis of 66 patients who had died at the Bicêtre, found that 55 had passed the fiftieth year and 34 had lived beyond sixty. The cause of death is usually some intercurrent affection not necessarily associated with tabes, though vesical inflammation and secondary infection of the kidneys are always to be apprehended.

Treatment.—In the management of tabes dorsalis it is well to keep in view exactly what may be accomplished. The retardation of the disease, or, better still, its complete arrest, constitutes a medical victory. Given the natural tendency of the disease to halt temporarily in occasional cases or to come to a permanent standstill, we must be chary of attributing too much importance to any line of medication. The intelligent purpose of treatment is: (1) To arrest any active syphilitic process that may be present; (2) to improve the general health; (3) to increase the nutritive condition of the cord; (4) to maintain as complete muscular control as possible, and (5) to meet the host of incidental disturbances as they arise.

While the rôle of *syphilis* in tabes is chiefly played in the past tense, it not rarely happens that active and manageable syphilitic lesions attend at least its early stages. Meningitis, myelitis, neuritis, and cerebral conditions or affections of the osseous system may declare themselves, and yield to antisyphilitics. It is not a bad plan to follow Erb in prescribing an active antiluetic treatment to clear the air; but it must be done with circumspection, as the cases are not infrequent in which mercury and the iodids are not only not well borne, but their use is productive of distinct harm. It is needless to insist on this course of medicine if it has been recently undergone in a thorough manner. At any rate, it is a good rule to give all syphilitics an annual course of mercury and iodids. Numerous French and German writers have of late strongly recommended intensive mercurial treatment with alleged good results in arresting or improving the disease. Intramuscular injections of soluble or insoluble preparations of mercury are used. Brockhart[2] thus treated 58 early cases, securing improvement in 33 and recession of the disease in 12. Lemoine, Devy, and Lerrede have had similar results.

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[1] "Sem. Méd.," 1903.

[2] "Monatsh. für praktische Dermatol.," 1902.

The author feels confident that he has arrested optic atrophy in several advanced cases by this method.

The *general health*, with all the conditions of hygiene, good air, and a proper diet that pertain to it, are worthy of painstaking attention. Not only does a good general state tend to retard the activity of tabes, but it protects the patient from the great dangers of intercurrent affections, especially of the acute variety.

The *local nutrition* of the cord and spinal apparatus may be improved by increasing its blood-supply. This may be mechanically effected by spinal stretching. It is not desirable to carry this out, as first was done, by hanging in the Sayre apparatus—a proceeding that is attended by a number of dangers. It can be effectively accomplished by having, after Benedict's suggestion, the patient fully flexed upon himself. Tourette and Chipault[1] have proven that the lower portion of the cord may actually be stretched by the forward bending of the trunk. Care must be exercised not to overstretch the patient at first, as the thigh- and back-muscles may easily be severely strained. Gradually, in the course of a week or two, through daily séances, the full body flexion, and consequent extension of the cord, can usually be attained without inconvenience. The flexed posture is to be maintained for two or three minutes only, and may be utilized night and morning. It often favorably modifies the lightning pains, and sometimes increases the sexual aptitude. The stretching is accomplished by seating the patient on a low table or on the floor, with the lower limbs extended. The head is then forcibly depressed toward the knees, which are not allowed to bend. The position is to be maintained not to exceed two minutes.

Local measures to the back, such as deep massage and very vigorous slapping, are of similar benefit to the deeper circulation. For this purpose a broad, stout piece of leather on a short handle may be used to vigorously flagellate the back. Its similar application to the soles of the feet and on other paresthetic or analgesic regions is useful. Counter-irritants along the spine have a certain value, perhaps only an insignificant one. The best application is by the thermocautery of Paquelin, repeated every seven or ten days. An intense white heat should be used, and small dots quickly and lightly made at intervals of an inch or two over the portion of the cord principally affected. They may with propriety be extended over the course of the nerves where the lightning pains discharge. Six, eight, or ten such cauterizations may be followed by two or three months' rest, and then repeated. Cold spinal douches, hot needle douches, or steam douches to the back are of comparable utility.

It is only of late that the value of *exercises and practice* to reëstablish coördination has been recognized. On the other hand, inactivity and disuse promptly accentuate the loss of muscular control. The purpose is not to accomplish feats of strength, and all strains and decided fatigue are to be sedulously avoided. Patients must be encouraged to faithfully and intelligently practise such movements, motions, and steps as are particularly uncertain. In this way they may sometimes be brought to stand and to walk with closed eyes after static ataxia has been well marked. Fränkel gives the following exercises, which are of two classes—those performed in and those performed out of bed, depending on the patient's helplessness:

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[1] "Nouv. Icon. de la Salpêtrière," June, 1897.

In bed, the patient is called upon to flex, extend, abduct, and adduct each leg separately and then both simultaneously. The knees and hips are likewise exercised. The patient is asked to place the heel of one foot on the big toe of the other foot. Place heel upon knee of the other leg and then slowly travel along the ridge of the tibia toward the ankle. Exercises are made alternately, first with one leg, then with the other, with open and with closed eyes. These exercises are attempted over and over again, with frequent rests. Patient is encouraged to persevere until he succeeds.

The exercises are repeated twice a day, a half hour in the morning and again a half hour in the afternoon.

1. Patient is placed with his back to a chair, heels together, then seats himself slowly in the chair, and is then made to rise in the same careful manner. No cane is used. If patient can not stand, an attendant is placed on either side to support him if necessary.
2. One leg is placed at an ordinary walking step in front of the other, and then placed with great exactness back into its original position. Same exercise is then performed with other leg. The patient, if necessary, supports himself by a cane or otherwise.
3. Walks three paces slowly and with precision.
4. Rest in standing position, one foot before the other; with hands placed akimbo he flexes his knees and slowly raises himself again.
5. Patient, as in exercise number 2, advances one foot, then returns it to its original position, and then places it one step behind the other. This exercise is usually a very difficult one, requiring, as it does, a great deal of balancing power.
6. Walk twenty steps as in exercise number 3.
7. Number 2 performed without a cane.
8. Stand without a cane, with feet placed together and hands on hips.
9. Stand without a cane, feet separated; various movements with the arms, grasping objects, forcing back outstretched hand of physician, etc.
10. Maintain same position as in number 9, flexing trunk forward, backward, to the right, and to the left.
11. Exercise number 9 with the feet together.
12. Exercise number 10 with feet together.
13. Walk along a painted line on the floor, patient supported by a cane.
14. Same without a cane.

Exercises for the fingers and arms are also employed, based on the above-mentioned principles.

These various exercises are to be progressively attempted and persevered in as coördinate strength improves. They may then be gone over again with closed eyes aided by a cane or assistant, then without aid. Fatigue, however, must be avoided. In advanced cases that have to rely on crutches, a tall "walking-frame" or roller-crutch, such as is used for children, may be employed. This gives support under the arms and enables the patient to exercise the legs. Precise, delicate motions with the fingers may also be developed in the same way. The employment of an intelligent masseur has given good results in some instances, as he can, by the use of graduated resistive movements, teach the patient precision in the use of his legs. The so-called home bicycle trainer has also given some assistance. On this the patient can pedal for a few minutes at a time, and the mechanism carries his feet and legs in definite curves.

In the pre-ataxic stage the patient should walk, stand, and balance on each foot, with closed eyes. Daily exercises of this simple character seem notably to retard the appearance of locomotor incoördination. Women are much less troubled than men in the control of the legs and feet, as a rule, and this may be due to wearing skirts, which prevents the use of the eyes in guiding the steps.

Regarding *internal medication* directed to the sclerosis, mercury, arsenic, silver, chlorid of gold, salts of zinc, strychnin, aconitin, atropin, and a multitude of others may be mentioned, but aside from some general tonic properties it is difficult to attribute any value to them. Ergot, first employed against a hypothetical chronic inflammation in which the sclerosis was supposed to consist, has proved itself of some value in controlling vesical disturbance, and against this feature of tabes may be employed, with precautions to avoid ergotism. Charcot's plan was to use it the first three days of every week, or it may be used on alternate weeks for one or two months, then a long interval and a repetition. It should be used in good-sized doses once or twice a day.

Against the *vesical weakness*, especially the sphincteric weakness, the method of Brandt to increase the strength of the pelvic floor is of service in tabes. The movements principally useful consist of having the patient separate and adduct the knees against resistance from two to twenty times twice daily, at the same time vigorously drawing in the pelvic floor and body outlets, and in massage of the perineal muscles.

For the *pains* of tabes and the *visceral crises* morphin is sometimes required, but the physician alone should administer it, to prevent the formation of a habit, and then only as a last resort. Ice, hot applications, sinapisms, and the coal-tar sedatives should be first thoroughly tried. Of the synthetic preparations, phenacetin seems the most efficient. Blisters and the cautery to the painful region and over the corresponding portion of the cord are sometimes promptly helpful, but must be used with circumspection as healing is often faulty.

Tabetic *joints* are best let alone. Nothing is to be gained by cutting operations, and very little by fixation apparatus except such as enable the patient to walk. Perforating *ulcer* is sometimes cured by stretching the nerve to the part.

Cystitis must be guarded against. If it develops, it must be carefully combated, and self-catheterization may have to be taught the patient. Urotropin in ten-grain doses, twice or thrice daily, may be employed for indefinite periods to keep the urine aseptic and prevent cystitis.

The *management of the individual* will often be found as difficult as that of the disease. He must not expect too much, but the physician must remember that he is human, and do what he can to encourage and cheer him in the face of his distressing affliction, and to insure his faithful attention to the numerous small and exacting details of treatment.

The appearance of any active syphilitic process demands prompt recurrence to specific treatment.

COMBINED SCLEROSES OF THE SPINAL CORD.

Occurring, perhaps, as frequently as tabes, many cases of organic cord-disease present symptoms referable to the simultaneous involvement of the lateral and the posterior columns, which are found sclerosed in widely varying proportion. This condition is termed *ataxic paraplegia* by Gowers, *progressive spastic ataxia* by Dana, and is known variously as *posterolateral sclerosis*, *combined posterior and lateral sclerosis*, and *combined tabes*, or sometimes as *spasmodic tabes*. Some cases show a tolerably definite limitation of the sclerosis, suggesting a systematic degeneration, but usually it is not strictly confined to the physiological tracts of the cord. For the most part the lesions are within the posterior half of the cord's cross-section, and are commonly embraced in the posterior arterial field (see p. 336 and Fig. 126). Occasionally the lesion also encroaches upon the peripheral portion of the cord in front, which belongs to the anterior arterial field. The symptoms are dominated by ataxia and spasticity, and the tendency is progressively toward paraplegic helplessness.

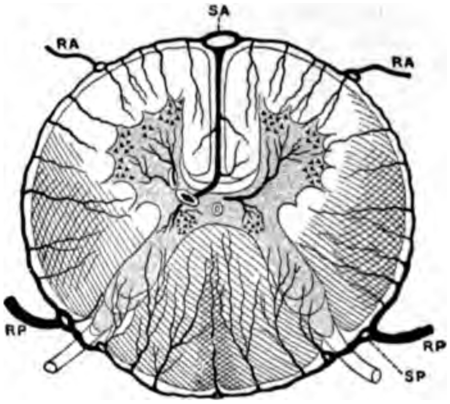

Fig. 196.- Combined posterolateral sclerosis. Scheme showing usual area of cord-change limited to the posterior arterial field of the cord (Brissaud).

Etiology.—The causes of combined sclerosis are numerous. It must be at once recognized that this extensive symptom group is frequently a secondary cord-process: (1) In a certain small number of cases, primarily tabetic, a diffuse myelitis also invades the lateral tracts; (2) it is found more frequently in general paresis than are lesions entirely confined to the posterior columns; (3) a diffuse myelitis gives rise to ascending and descending degenerations that furnish a posterolateral sclerosis; (4) leptomeningitis may entail a marginal myelitis that invades the periphery of the cord and principally affects its posterior half; (5) vascular lesions, affecting principally the posterior field, induce a sclerosis in the posterior and lateral tracts; (6) pellagra sometimes causes a combined posterolateral sclerosis; (7) toxic conditions, as in the pernicious anemias, may cause it; (8) the posterolateral sclerosis may be evidence of an embryonic deficiency as in Friedreich's ataxia.

There is frequently a history of antecedent syphilis or one of exposure to cold, traumatism, concussion, or muscular strains. Sometimes acute infections lead to it. It is much more common in males than in females, and usually appears between twenty-five and forty years of age.

Morbid Anatomy.—The sclerosis implicates both the lateral and posterior columns, but sometimes one, sometimes the other with greater intensity. The *columns of Goll*, usually commencing above the lumbar enlargement, are degenerated to the medulla. The *columns of Burdach* are also affected mainly above the lumbar enlargement, but less intensely, and the root-zone usually escapes. The *crossed pyramidal tract* is, as a rule, partially affected, its inner portion being customarily spared. Its lesion is slight in the cervical region, and increases in the lower dorsal and lumbar portions of the cord. The *direct pyramidal tract* is only affected when the lesions are intense in the upper cervical region, and even then but slightly. The *direct cerebellar tract* is always intensely affected. The ascending *tract of Gowers* sometimes escapes, sometimes is partially affected. The *gray substance* is usually intact. Rarely the anterior cornual cells or those of the posterior horn or of Clarke's column may be slightly modified.

The sclerotic areas differ from the degenerated portions of a tabetic cord in presenting hypertrophied axes-cylinders, many spider-cells, and notable changes in the blood-vessels. The alteration in the nervous tissue is also proportionate to the vascular lesions and most intense in their neighborhood. Occasionally a chronic meningitis is present. Cranial nuclear involvement has been found in some cases.

To sum up, the lesion is of a myelitic character rather than that of a pure parenchymatous Wallerian degeneration. It affects the posterior columns from the lower dorsal area upward and the lateral tracts from the cervical region downward in increasing intensity, precisely as do secondary degenerations. The lesion is indiscriminate in localization and bears a close relation to the arterial supply of the part.

Symptoms.—The symptoms of the combined scleroses are those of spastic paraplegia and locomotor ataxia variously combined and associated. One case shows a preponderance of ataxia, another of spasticity, depending upon the distribution and intensity of the lesion in the posterior or lateral columns and upon its vertical extent. Lesions of these tracts give us, on the part of the posterior columns, incoördination, an ataxic gait, Romberg's sign, diminished or lost reflexes, lancinating pains, anesthesias and paresthesias, ocular symptoms, vesical and genital derangements, etc. Related to the lesion of the lateral tracts we have exaggeration of tendon reflexes, foot-clonus, motor enfeeblement, cramps, spasms, and the spastic gait. By their association in posterolateral sclerosis we usually find ataxia and spastic weakness, with increased tendon reflexes, well marked in the lower limbs, less pronounced in the upper extremities, and cranial-nerve symptoms are seldom entirely absent.

The *onset* of the disease, when not due to a diffuse myelitis of more or less acute character, is insidious. One of the earliest complaints is that of becoming readily tired in walking, the legs feeling heavy and weak. Some *ataxia* soon presents itself, as is shown by the unstable station with closed eyes, or difficulty in turning or walking under the same condition. The *knee-jerks* will be found increased in activity and amplitude. The *cutaneous reflexes* may be increased or diminished. The *cremaster* is usually inactive. *Muscular weakness* can be easily detected. The muscles do not lose their contours and are often firm to the touch, but are incapable of strong contraction. The ataxia increases; the gait becomes very uncertain, with a tendency to sprawling, and at the same time it is spastic, so that the feet are not readily brought forward, and they may be shaken by *clonus*, which is often easily developed in the calf and rectus femoris muscles. Some *vesical weakness* is very commonly encountered, and loss of sexual appetite is frequent, though erections and even painful priapism may annoy the patient.

Sensory symptoms are much less common, and when present are extremely slight as compared with tabes. Instead of lightning pains we find *dull aches*, usually in the thighs and small of the back and over the sacrum. *Paresthetic sensations* are not uncommon, but actual anesthesia or any considerable blunting of tactile sensation is very rare. The *girdle pain* is occasionally present. Visceral *crises* are not common, and are not severe when they do occur. Rectal and vesical *tenesmus* and painful *cramps* in the legs at night are sometimes the source of much complaint, and the corresponding *sphincters* may be impaired in exceptional cases. There is often inability to satisfactorily empty the bladder and bowels. Urinary *retention* may then lead to *cystitis*.

The upper extremities almost invariably show an exaggeration of the tendon reflexes, and some rigidity and weakness is not infrequent. An exaggerated *jaw-jerk* may be easily demonstrated in most instances. Tremulous *twitchings* in the tongue and face are not rare, and speech may be thickened slightly, even in the cases not associated with paretic dementia. *Pupillary symptoms* are not uncommon, but the light-reflex is rarely lost when accommodative contraction persists. Inequalities of the pupils, irregularities of outline, and sluggishness of motility to various stimuli are frequently noted. The *mental state* is ordinarily normal, but there may be some loss of memory, dependent, mainly, on lack of concentration and attention. Prolonged mental application is usually impossible. In short, it is a neurasthenic state.

As the disease progresses the paresis in the legs and the spasticity increase. The static ataxia is also exaggerated actually and also by the increased weakness. Finally, the patient is unable to walk and the spastic ataxia becomes marked in the arms. The paraplegic state is thus induced, but sensory disturbance, as a rule, is insignificant to the last.

Course.—While the disease is *progressive* it is much less rapid in its course than tabes, and the great majority of cases never become entirely unable to walk. Of itself it rarely causes death. Bedsores, cystitis, and kidney disease are the chief dangers. Intercurrent affections necessarily find victims in these devitalized subjects. The cases that commence as tabes or as myelitis present features referable to their origin. Early loss of knee-jerks, the presence of girdle pains, anesthesias, analgesias, or joint-lesions with spastic features in the upper spinal levels are probably due to myelitic extension from a posterior sclerosis. Those presenting rapid onset and girdle features and localized atrophies are usually attributable to diffuse myelitis.

Diagnosis.—The diagnosis of progressive spastic ataxia, which is the best descriptive name for this symptom group, depends upon the muscular weakness, the ataxia, the spasticity, and the progressiveness of the disease. In early stages when the ataxia is prominent it is usually mistaken for *tabes*, but the muscular weakness, the increased reflexes, the insignificant sensory disturbance, the absence of girdle pains, and visceral crises should at once differentiate it. In later stages when the paraplegic features are preëminent the ataxia is lost in the motor extinction, but a history of it can usually be obtained. Cross or *focal lesions* are now sometimes thought of. The sensory integrity and the presence of symptoms in the arms and in the cranial nerves should serve to exclude such limited lesions. The *family ataxias* present a somewhat similar symptomatology, and the cord-lesion has about the same distribution. The familial features, the nystagmus and articular disturbance, its occurrence in early life, and in the type of Friedreich the absent patellar reflex, should make the diagnosis plain. *Cerebellar tumor* can induce ataxia, increased knee-jerks, and weakness in the legs, but also usually has vomiting, occipital pain, choked disc, and retracted head, and may have forced attitudes or movements to distinguish it.

Prognosis.—The slowly progressive tendency of the disease has been sufficiently insisted upon. Stationary periods, or those comparatively so, are very common, and even improvement in the individual features of some cases are not infrequent. Complete relief, in the nature of things, is an impossibility, and the onward course of the paretic and spastic features is taken up sooner or later. Mental disturbance, while uncommon, may appear and paretic dementia ensue. Every case is to be judged by itself. The rapidity or slowness of development in the early years of the malady is likely to mark it throughout. As already indicated, only a small portion of the cases reach absolute helplessness.

Treatment.—The treatment of a case of progressive spastic ataxia will be modified by its variety and origin. When secondary to tabes, the treatment is the same as in that disease. The history of syphilis is even a stronger indication for antiluetic treatment than in tabes, as syphilitic activity may directly influence the condition of the cord and be amenable to proper medication. Counterirritation over the spine, spinal stretching, and exercises to reëstablish coördinate motor control are valuable. Thermic baths, and hydrotherapy generally, here find a very useful application. As a rule, the use of hot douches, sprays, and baths lessen the spasticity, but in rare cases cold to the back has the better effect. Massage of the muscles and graduated exercises are important, but the fatigue point should be carefully estimated and never exceeded. To this end the paretic features are the guide, and the sensation of fatigue the criterion. Owing to the hyperexcitability of the reflexes, massage sometimes is not well borne. General measures looking to the physical health and the mental quietude of these patients add to their comfort and to the length of life. The bladder condition should receive constant watchfulness, as a high degree of retention may develop without attracting the patient's notice.

COMBINED CORD-LESIONS IN ANEMIAS AND CACHEXIAS.

Nonne was the first to demonstrate decided changes of the spinal cord in cases of pernicious anemia. Somewhat similar changes in the retina were well known, and apparently identical degenerations take place in the brain. This observer found degenerative changes in the cords of ten out of seventeen cases of pernicious anemia though symptoms referable to the cord had been observed in but two. The changes are principally located in the white matter of the cord, and show a decided tendency to mainly affect the posterior half, giving rise to a group of symptoms referable to the posterior and lateral tracts. In some instances, however, the anterior portion of the cord and even the gray matter is involved. By experimentally induced anemia in the brain and cord Massaro, Scicilliano, and Soukhanoff have demonstrated similar cellular changes in animals. Teichmueller, Minnich, Lichtheim, in Germany, Russell, Batten, Collier, in England, Dana, Putnam, Riggs, and Billings, in this country, among many others, have not only confirmed Nonne's findings but broadened the general field of degenerative conditions secondary to depraved physical states. The consensus of opinion now seems to be that the cord changes are due to a toxic process, secondary to many seriously debilitating systemic conditions, acting upon a nervous system of deficient attributes, shown either by a marked neurotic heredity or by general physical defects.[1] The operation of the toxic process is apparently by way of the vascular apparatus, giving rise to hemorrhagic or interstitial changes resulting in more or less sclerotic transformation.

Women are affected nearly three times as frequently as men.

Symptoms usually appear after the age of thirty, most commonly in the fourth decade of life. In addition to the features of the anemias and cachexias and the conditions arising from these states, there is often a decided mental irritability. Atrophy of the optic nerve has been observed in some instances. Epileptiform attacks have occurred.

The symptoms referable to the spinal cord are those of ataxia, spasticity, and weakness, variously combined, giving rise, as a rule, to an ataxic paraplegia similar to that described in the foregoing section. Early in the disorder the tendency is to increased reflexes, which later tend to subside or may entirely disappear or persist. In some cases the knee-jerk is lost early. Girdle sensations are not uncommon. Paresthesiæ occur from the first and are persistent. Neuralgias are common, so that multiple neuritis is often suspected. Bed-sores, cystitis, and sphincteric palsy may be encountered in terminal states exceptionally.

The **duration** of the disorder is variable. Many cases reach a profound degree of exhaustion before the cord symptoms develop, in which case they are likely to rapidly attain a severe grade. In other instances paresthesia, increased reflexes, and motor weakness come on insidiously and endure for a number of years, the general physical state being relatively but little impaired. In a general way these cases last from one to five years, the tendency being steadily for the worse, though slight remissions may be encountered. In some instances the paralytic and sensory disturbance becomes rather rapidly greater in the lower extremities and steadily mounts upward like an ascending myelitis or

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[1] Putnam and Taylor, "Jour. of Ment. and Nerv. Dis.," Jan., 1901.

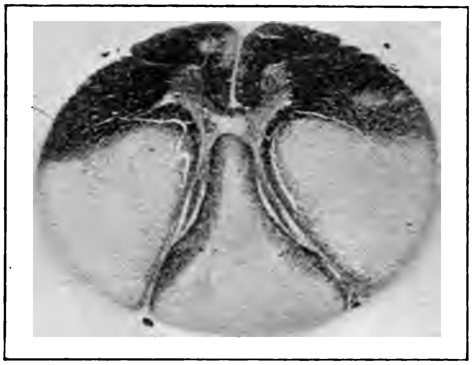

Fig. 197.

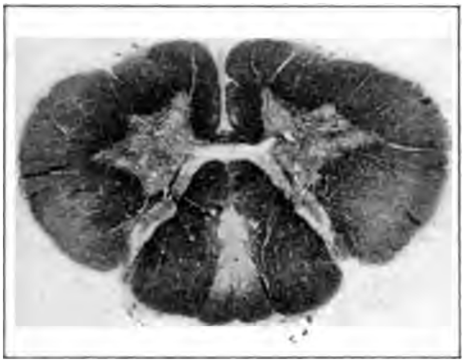

Fig. 198.

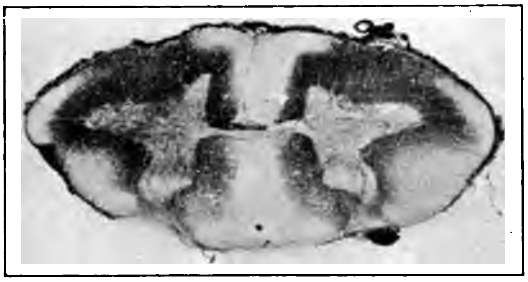

Fig. 199.

Figs. 197, 198, and 199.—Sections from three spinal cords showing degenerations due to pernicious anemia (Billings).

may advance and recede many times. The ultimate outcome is almost invariably fatal, and the **prognosis** depends mainly upon the systemic state.

The **treatment** is that of the anemia or underlying cachexia, the secondary toxic conditions, and the resulting changes in the cord. The disabilities arising from involvement of the cord have to be met by gymnastics, exercises, and massage.

FAMILY ATAXIA.

In 1861 Friedreich reported several cases of ataxia occurring in the children of one family. They presented, among many symptoms, the loss of the patellar reflex, incoördination of all four extremities, nystagmus, disturbance of articulation, and a progressive tendency to helplessness. Similar cases have since been reported in every country, until hundreds may be collected from the literature. In some instances the disease is found to appear in several generations, but, ordinarily, it is confined to a single family, and usually appears before adolescence. To the symptom group presented by these cases the name of *Friedreich's disease*, or *Friedreich's ataxia*, has been given.

In 1880 Fraser[1] reported a series of cases extending through several generations, marked by the same symptoms as occur in Friedreich's group, excepting that the reflexes were increased and ankle-clonus sometimes present. Nonne, in 1891, reported a similar family, and clearly pointed out the features in which the cases falling under his observation differed from the Friedreich variety, and especially the fact that they developed later in life,—namely, during or after adolescence,—and that they frequently showed visual defects and optic atrophy. Early examinations of Friedreich's form discovered spinal sclerosis, embracing the posterolateral area throughout the cord, and a cord of diminutive size in some instances. To this, in some cases, is added an undersized cerebellum and cranial-nerve lesions. In one of Nonne's cases only an extreme smallness of the cerebellum and cord was found. Switalski[2] reports a case showing diminutive proportions in cord and cerebellum, degenerations of Goll's columns, the direct cerebellar tract, Gowers' tract and some atrophy of the anterior horn cells. The cerebellum showed a diminished number of convolutions, separated by deep fissures, and a paucity of white substance. A family very similar to those of Fraser and Nonne was reported by Sanger Brown,[3] and autopsy on one of his cases showed no gross cerebellar defect.[4] Transition cases[5] are being observed that furnish every intermediate variety between the spinal cases on the one hand and the cerebellar cases on the other. For the latter group Marie has used the designation *pseudoataxie cerebelleuse*, and *hereditary cerebellar ataxia*. Senator[6] sees in Friedreich's ataxia only the manifestations of teratological cerebellar and spinal defect. In the cases of Nonne and others marked by cerebellar atrophy, or, more probably, cerebellar agenesis, the same origin is apparent, and the variety of clinical cases and anatomical findings seems to depend upon the location of the principal defect. The familial and hereditary features of these cases at once declare their embryonic character and origin. The portions of the cord that undergo sclerotic changes are precisely those which are last to develop and become myelinated, only reaching completion at the end of the ninth or in the tenth month.

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[1] "Glasgow Med. Jour.," 1880.

[2] "Nouv. Icon. de la Salpêt.," Sept., 1901.

[3] "Brain," 1892.

[4] "Brain," 1897.

[5] Haushalter, "Rev. de Med.," 1895.

[6] "Berlin. klin. Wochens.," 1893.

Etiology.—The most important etiological feature of the family ataxias is their embryological nature. Why a mother should give birth to several children with defective nervous structures or other teratological defects it is impossible to say, though phthisis and other exhausting cachexiæ have presented in some of these parents. As is common in familial nervous diseases, there is a tendency to a preponderance of males, and the transmission is usually by the female line. Both of these points are illustrated in the genealogical diagram of Brown's cases (Fig. 200). In a given family there is frequently noted a tendency for

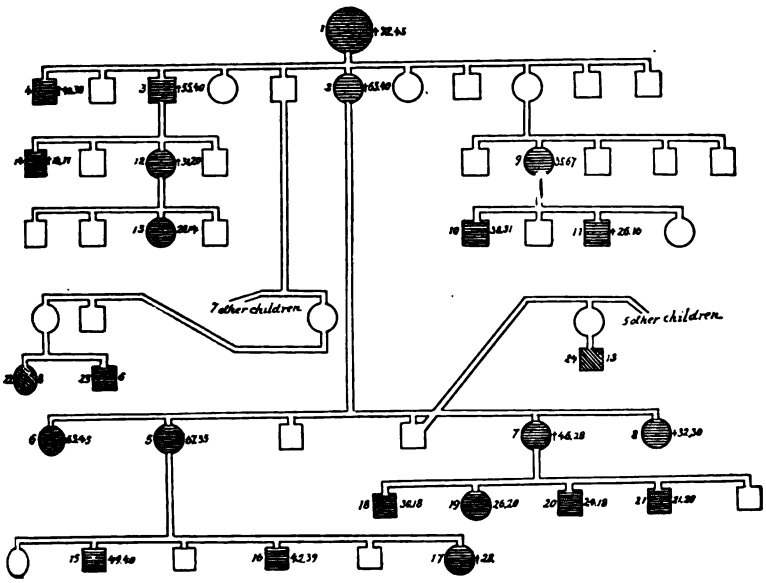

Fig. 200.—Family tree of hereditary ataxia, reported by Dr. Sanger Brown. Explanation of diagram: Shaded inclosures indicate hereditary ataxia. Squares indicate males; circles females. The numbers to the left refer to the cases in Brown's paper; the first number to the right, the age at death or the present age. † indicates deceased. The last number indicates the age at onset.

the disease to be manifest at a progressively earlier age in successive children. The symptoms are likely to appear at the developmental periods of life. In some cases they are congenitally evident; in others the first or second dentition, the ages of puberty, adolescence, and complete adult sexual differentiation and reproductive ability seem to make demands that the defective neural apparatus can not meet, and thereafter retrogrades. In exceptional cases the disease appears late in life. Friedreich's type usually occurs before the age of fourteen; hereditary cerebellar ataxia, or Marie's form, after pubescence. In many cases, especially in children, and, therefore, usually in Friedreich's variety, the occurrence of an infectious fever appears to precipitate the symptoms of the disease. In these cases, too, the cord-lesions correspond to those of the combined scleroses and occupy the posterior arterial field of the cord's cross-section. The embryonic vulnerability of this portion of the cord may serve to locate the lesions in these instances in the lower levels through the intermediary of the vascular supply and the action of toxic factors.

Morbid Anatomy.—The morbid anatomy of these family ataxias varies as the case corresponds to the spinal or the cerebellar type. The cases that conform strictly to *Friedreich's syndrome* show a *posterolateral sclerosis* analogous in distribution to that of progressive spastic ataxia. There is a decided sclerosis of the *columns of Goll and Burdach*, with shrinking of the cord in this region throughout its entire length. There is sclerosis of the *crossed pyramidal tract*, of *Gowers' tract*, of the *direct cerebellar tract*, of *Lissauer's tract*, and frequently atrophy of the cells of *Clarke's column*. The *posterior horn* and its cells are shrunken and in rare cases the anterior cornual cells are degenerated. Marie insists that the changes in the pyramidal tract are confined to the fibers related to the direct cerebellar tracts and Gowers' tracts, and do not directly affect the upper motor neurons. The portions of the cord that last develop are thus affected by the retrogressive changes of the malady. As a rule, the *cord is undersized* and may present only two-thirds of its usual thickness. In a few cases, notably that of Menzel, the *medulla* and *cerebellum* were also reduced in size and degeneration has been traced into the *cerebrum*. The *posterior roots* and *ganglia* are usually normal. Rennie,[1] however, found degeneration of posterior root-fibers and posterior root-zones. The cranial nerves are generally spared in the spinal variety, but the hypoglossus and optic nerves have been found diseased.

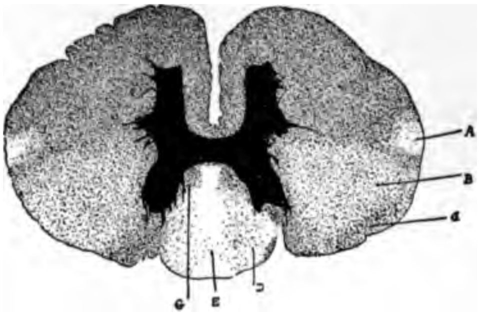

Fig. 201.—Dorsal cord-section in Friedreich's disease, showing distribution and relative intensity of sclerotic changes (Marie).

The *cerebellar form*, in the few cases on record, has presented atrophy of the cerebellum, as in Fraser's case, in one of Nonne's, in Switalski's, etc. This is absent in Brown's cases, but the cerebellar tracts in the cord were involved. This cerebellar atrophy is said not to be sclerotic. The gray substance is abnormally thin, Purkinje's cells are few in number and undersized, the white matter is less voluminous than usual, and the weight of the organ is reduced a third or a half. The cord in these cases of Marie's type is undersized, but shows no sclerotic degenerations. There appears to be a pure genetic poverty of fibers, or else they have disappeared, leaving no trace. In Brown's cases, as reported by Meyer and later by Barker,[1] there was degeneration in the dorsolateral cerebellar tract and degeneration in the gray and white matter of the cord, medulla, and cerebellum. Optic atrophy is not infrequent.

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[1] "Br. Med. Jour.," July 15, 1899.

Symptoms.—The *motor symptoms* are the most prominent. They commence with clumsiness, especially in walking. The child stumbles over every trifle and becomes more and more unsteady on its feet. Later the *gait* is staggering and drunken in its uncertainty. *Ataxia* marks every movement and position, but there is no rigidity even in the cases presenting increased reflexes. All movements are clearly intentional, though clumsily executed, and have for their purpose to maintain the insecure equilibrium. *In standing* the body is constantly swaying, and shuffling steps in various directions are frequently taken to maintain the balance. With closed eyes the difficulties of the station and gait are not notably increased, and there is practically no loss of the *muscular sense*, only an ataxic incoördination in executing movements. Frequently coarse, rhythmic, *trembling* or *jerky movements* are made on attempts to use the extremities, and at rest often a finger or the wrist, shoulder, elbow, head, or lower extremity is moved or twitched in a spasmodic manner. Such movements are frequently seen in the face, especially when the patient begins to talk or when some emotional expression is called out. Ordinarily, complete support of the part or its complete rest causes them to cease. The head, in a similar way, nods in one direction or another, and may, in late cases, roll around on the shoulders as if articulated with ball and socket. As the patient sits, the body may also sway about above the hips. An examination of the *muscular strength* finds it but slightly reduced, and clearly indicates that there is no serious paralytic state present. The muscles do not waste except in cases of long standing, and then only rarely and mainly about the shoulders and hands.

The *sensory symptoms* are objectively and subjectively very slight or entirely wanting. A few cases have pains or even darting pains in the early stages. The presence of anesthesia or analgesia should raise a suspicion of hysteria. The muscular sense is practically perfect.

The *superficial reflexes* are not disturbed. In cases of the Friedreich type the deep reflexes are diminished or abolished and the knee-jerk usually disappears very early. In Marie's form the deep reflexes are exaggerated and foot- and rectus-clonus are commonly encountered. The *sphincters* are unaffected.

The *face* presents an appearance of hebetude that deepens as the disease advances and in some instances first calls attention to its invasion. The features droop in a mask-like blankness of expression, often intensified by a half-open mouth. In emotional expression the ataxic unbalance appears, resulting in contortions, or, at least, exaggerations of facial movements. There is usually well-marked *nystagmus*. The oscillations of the globe, however, tend to cease if the eye is allowed to remain at rest, and vary in amplitude and are increased by effort, like all the other manifestations of incoördinate muscular balance. It is often necessary to direct the eyes widely from the direct line of forward vision to develop the nystagmic jerking. This can usually be accomplished by having the patient fix his eyes upon an object in an outward and upward direction. Palsies of the ocular muscles are extremely rare. Joffroy has published one case, and another is illustrated here. In Friedreich's form *optic atrophy* is a rare exception. Small [1] reports three out of four cases in one family showing some degree of it. In Marie's form, on the other hand, it is a common finding, and changes in the visual *acuity* and in the form- and color-*fields* are frequently encountered. The *pupillary reflex* is usually normal in Friedreich's form, but may be affected in Marie's group.

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[1] "Decennial Pub., University of Chicago," 1903.

The *speech* is ataxic, if the expression may be used. *Modulation* of the voice and prompt *enunciation* are defeated by the tardy and incoördinate action of the muscular apparatus. It reminds one of the gait; some words come quickly, others slowly, and the voice tones change suddenly and irregularly in pitch or other qualities, though tending to a monotone. The *intelligence* of these patients is not much impaired in the early years of their malady, and is often belied by their apathetic and empty faces, but in the later stages of the disease the mind is frequently sluggish or shows some retardation of development. In Friedreich's form physical development is also frequently retarded if the disease manifests itself before puberty, and sexual functions are correspondingly backward.

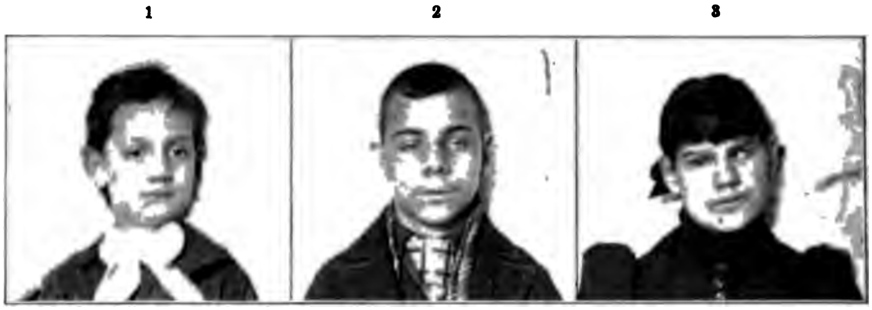

Fig. 202.—Apathetic facies in Friedreich's ataxia. 1, O. F., eight years old, affected one year; 2, F. F., seventeen years old, affected three years; 3, R. F., twenty years old, affected five years.

Trophic and vasomotor defects are very rarely encountered, or are entirely lacking except a peculiar conformation of the feet and a slight vertebral scoliosis that are frequent in Friedreich's form. The *club-foot* consists of an exaggeration of the plantar arch which shortens the foot notably and causes the toes to assume the "hammer" position, extended at the first and flexed at the other phalanges. This is particularly marked in the big toe, which usually is first affected and is sometimes the first symptom of the disease detected by relatives conversant with its forms. The deformity is increased when the foot is extended upon the leg, and if slightly marked, may disappear when the patient is standing. A similar deformity of the foot is also encountered in some of the family and sporadic cases of progressive muscular atrophy. The *scoliosis* appears in most of the well-developed cases of Friedreich's form, and occupies the dorsal and lumbar spine. It is usually slight.

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[1] "Med. Record," N. Y., July 20, 1895.

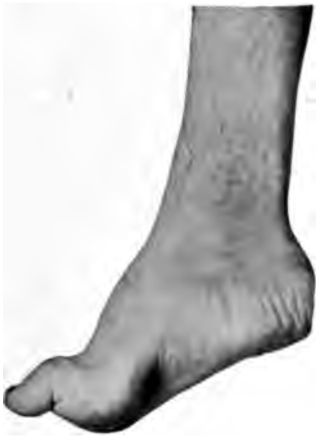

Fig. 203.—Clubbed foot of Friedreich's disease, showing shortened arch and retracted great toe.

Varieties.—The two major varieties making up the group of family ataxias have been indicated throughout the preceding pages, and there are many authoritative writers who regard them as distinct entities. Their principal differences are contained in the following table. The cerebellar features clinically predominate in both and autopsies are yet too few to justify their complete separation, especially as all intermediate varieties are being reported:

FRIEDREICH'S FORM.	MARIE'S FORM.
Hereditary spinal ataxia appears usually before puberty.	Hereditary cerebellar ataxia appears usually after puberty.
Choreic movements in upper extremities and oscillations in head and trunk frequent.	Very common and pronounced.
Optic atrophy and amblyopia very exceptional.	Common.
Tendon reflexes diminished or absent.	Increased, foot-clonus frequent.
Club-foot and scoliosis common.	Exceptional.

Course.—The first symptoms to attract attention, unless the case is congenital, are the difficulties in walking. Sometimes certain families, who have learned to know the symptoms from the number of cases among them, recognize, by the facies or the scoliosis or the club-foot and cramped toes, that another member has been added to the list. In the course of three to four years the ataxia in all four extremities is well marked. It advances and augments and in another similar period takes the patient off his feet and renders him practically helpless. In this state he may live ten to twenty years and finally die from intercurrent disease. While the course of the disease is commonly steadily progressive, long remissions may occur and sudden aggravations take place, but death does not result directly from the disorder.

Diagnosis.—The diagnosis of a case of family ataxia is almost self-evident if numerous members of the same or succeeding generations are affected, but in certain instances no such collateral cases exist. The differential diagnosis must then be made from tabes, chorea, and insular sclerosis. In *tabes* the gait lacks the cerebellar quality. Posterior sclerosis also presents visceral crises, lightning pains, vesical weakness, sensory disturbances, and pupillary symptoms. It is a disease of full adult life, has commonly a syphilitic history, and nystagmus is extremely uncommon in it. The cases of family ataxia occurring late in life usually present increased reflexes.

From *chorea*, for which it is frequently mistaken, especially in children, family ataxia may be distinguished by the major affection of the upper extremities in Sydenham's disease, its rather abrupt onset, and the absence of nystagmus, scoliosis, club-foot, and persistent abnormalities of the tendon reflexes. Huntingdon's family chorea of adult life has its mental features and forced attitudes and movements, which are greatly in excess of anything seen in the family ataxias.

Insular sclerosis presents the closest resemblance to family ataxia, especially Marie's form; and as it also may affect several members of the same family, the differentiation may be most difficult, if not impossible. We must recall its distinctive intention tremor, its lack of static instability, its scanning staccato speech, its spastic features and gait.

Prognosis.—The outlook in these family ataxias is always gloomy. Aside from the helplessness there is no suffering, and, as a rule, they bear their lot with an apathetic good nature.

Treatment.—The management of these cases does not promise much improvement. Some have thought that spinal stretching and electricity improved the incoördination. Carefully planned exercises may, perhaps, assist in the same direction.

HEREDITARY SPASTIC PARAPLEGIA.

Commencing with Strümpell, groups of cases have been recorded by Bernhardt, Philips, South, Newmark,[1] Achard and Fresson,[2] Bayley,[3] and others, which present pure spasticity and familial traits. Bayley traced the disease through five generations, the individuals affected showing marked similarity in all respects. In his series it seemed that the disease once escaped did not reappear in the descendants. Spiller[4] reports a family showing the disease in eight generations.

The spastic condition may appear at any age from the first (Achard and Fresson) to the fifty-sixth year (Strümpell) and very commonly is preceded by some infectious disease, as typhoid, measles, etc. There is paresis only in the terminal stages and no ataxia. The muscular hypertonus and increased reflexes are alone noted. Sensory and sphincteric difficulties are absent and cerebral features are insignificant, or only such as are attributable to defects in the intracerebral portion of the motor pathway. The leg rigidity may, however, render the gait markedly spastic and walking finally impossible.

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[1] "Med. News," Feb. 16, 1897. [2] "Gaz. hebdom. de Med et de Chir.," Dec., 1896.
[3] "Jour. Nerv. and Ment. Dis.," Nov., 1897. [4] "Phila. Med. Jour.," June 21, 1902.

The **symptoms** are referable to the pyramidal tracts which, with the columns of Goll and the direct cerebellar tracts, were found degenerated by Strümpell. Erb,[1] who called it a primary spastic paraplegia, has collected ten postmortem reports showing the sclerosis to be mainly confined to the lateral portions of the cord and principally to the crossed pyramidal tract. Jendrassek [2] believes that the process may begin in the brain, bulb, or cord, and is introgressive in nature. From the familial feature of these cases it is evident that the basis of the paraplegia is a teratological defect and its sometimes late appearance is no bar to such belief.

Treatment promises little, though massage and carefully selected exercises may accomplish something.

The **prognosis** is not hopeful, but the course of the disease is usually very slow and for a given family nearly the same in all the affected members.

SCLEROSIS OF THE CORD DUE TO VEGETABLE INTOXICANTS—ERGOTISM, LATHYRISM, PELLAGRA.

Ergotism.—Epidemics of ergotism have arisen in various localities of the Old World, usually in times of bad harvest, when the people were reduced to eating ergoted grain. Brain symptoms in the form of mania are often associated, and epileptoid attacks are common. The disease respects neither age nor sex, and sometimes animals present similar cord symptoms. These, in man, consist of paresthesias, lightning pains, girdle sensations, analgesia, static instability, and an ataxic gait with obliteration of the patellar reflexes. Neither optic atrophy nor pupillary stasis occurs. In all recent autopsies a sclerosis confined to the posterior columns, and conforming closely to the changes in tabes, has been found. In many instances the symptoms develop after the poisonous food has been discontinued for some months. The tendency of the disease is toward recovery by progressive amelioration during several years. In some instances even the knee-jerk is restored. The mortality, however, in acute cases, is rather great. (For fuller particulars the reader is referred to the article of Tuczek, "Archiv f. Psychiatrie," Bd. xiii, S. 99.)

Lathyrism.[3]—Epidemics of this disease in Europe, Africa, and Asia have been noted for centuries. The cases are marked by rigidity of the lower extremities coming on within a few days, and paraplegic symptoms, with weakness of the bladder, generally ending in slow recovery. They coincide with famine conditions, when the people eke out their subsistence with the various vetches, such as *Lathyrus sativus*, or *cicera*, or *clymenum*. The morbific constituent appears to be an alkaloid not destroyed by ordinary cooking. The symptoms come on gradually or sometimes rather abruptly, and a spasmodic paraplegia is developed, producing the paraplegic state, so far as motion and locomotion is concerned. Sensation is not materially disturbed, though paresthesias and some blunting of cutaneous sensation are usual. Mental symptoms and cerebral features, including involvement of cranial nerves, seem to be lacking. The exact lesion is not known, but the symptoms all point to disturbance in the lateral tracts, or, perhaps, to a posterolateral sclerosis with preponderating spasticity. Often such cases suggest a focal lesion of the cord, but the absence of serious sensory disturbance and the complete recovery that, as a rule, ensues are not consistent with such a point of view.

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[1] "Br. Med. Jour.," Oct. 11, 1902.

[2] "Deut. Arch. f. klin. Med.," Bd. lviii.

[3] Brunelli, "Trans. Seventh Internat. Cong.," vol. ii, p. 45.

Pellagra [1] is sometimes confounded with ergotism. It has occurred mainly in poverty-stricken Italian districts, and is traceable to the use of diseased and fermenting maize. It produces mental symptoms that usually call for asylum treatment. In an acute form, called pellagrous typhus, it may rapidly cause death, and then myelitis, enteritis, meningitis, and lymphatic adenitis plainly declare the toxemic nature of the malady. Many cases are marked by a rough, intense, rapidly extending erythema, usually commencing about the neck. Belmondo considers the disease to be due to a specific organism. Lombroso attributes it to a fungus in the maize.

The cord-lesions that are found consist of a leptomeningitis, often with much thickening, and even with the formation of osseous plaques. In the cord itself the anterior cornual cells are frequently atrophied and pigmented. There is commonly a posterolateral sclerosis. This affects the colums of Goll and Burdach, mainly in the upper cord-levels, but spares the root-zone of the postero-external column. The crossed pyramidal tract, especially the lower portion, is also sharply sclerosed, the direct cerebellar tract usually escaping.

The spinal symptoms correspond. Ataxia is most marked in the upper extremities; spasticity is pronounced in the lower limbs. The iridian reflexes are spared, and cutaneous sensibility is not much affected. Strangely, in spite of the usual changes in the anterior cornual cells, muscular atrophy is insignificant. The disease, clinically and anatomically, presents much resemblance to paretic dementia.

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[1] Tuczek, "Monographie sur la Pellagre," 1893.

PART VI.

DISEASES OF THE GENERAL NERVOUS SYSTEM WITH KNOWN ANATOMICAL BASIS.

AMONG the various diseases that have already been described, several have presented symptoms referable to the various portions of the entire nervous apparatus and properly belong to this section. Thus, in *multiple neuritis* all nervous levels may be invaded. Cerebral, spinal, and nerve-trunk lesions are all encountered. The same is true of *cerebrospinal meningitis*, *tabes*, *paretic dementia*, *bulbospinal muscular atrophy*, *ergotism*, *lathyrism*, *pellagra*, and *polioencephalomyelitis*; but for purposes of comparison as well as contrast it was thought most expedient to take them up in the order chosen, being guided by the preponderance of nerve-trunk or spinal-cord features in each instance. To those named we here add *multiple insular sclerosis* and *syphilis of the nervous system*. With some of the syphilitic lesions we are already acquainted.

CHAPTER I.

MULTIPLE CEREBROSPINAL SCLEROSIS.

Multiple cerebrospinal sclerosis—*multiple sclerosis*, *insular sclerosis*, *sclerose en plaques*, or *disseminated sclerosis*—is a disease secondary usually to some intoxication or infection often of a mixed sort. It is marked by numerous islets or plaques of sclerosis irregularly distributed in the brain, cord, and cranial nerves. These are related to the blood-supply of the part and probably primarily vascular.

Etiology.—This sclerotic disease is not very common. For instance, Uhthoff in six or seven years could only gather about 100 cases from all the hospitals and clinics of Berlin. It affects both *sexes* indifferently and presents a large preponderance of cases between the *ages* of twenty and thirty years. Marie asserts its absolute rarity after forty, but cases do occur. Children are sometimes affected and it may even be congenital. Considerable stress was formerly laid upon *heredity*, but this element is chiefly manifest in a neuropathic tendency. Erb, Oppenheim, and Duchenne have cited examples of direct succession, and it has, in a small number of instances, affected several children of the same family. Hervouet has even seen nine cases in one generation. *Overwork*, *cold*, *traumatism*, and various *excesses* have been accused as causative. Such relation is subject to doubt, though all these influences are capable of aggravating the disease when once established and of precipitating additional manifestations of its activity. Hoffman,[1] for instance, in a study of 100 cases, attributed the cause to trauma in 13, but in one-half of the cases no alleged cause could be mentioned. The most important etiological factors are the *infections*. Typhoid, pneumonia, malaria, measles, scarlet fever, small-pox, diphtheria, whooping-cough, erysipelas, dysentery, cholera, influenza, and the puerperium have been followed by the development of the symptoms of multiple sclerosis within a few weeks or months.[2] Oppenheim [3] found that in eleven out of twenty-eight cases treated by him the patients had long been exposed to the influence of lead, copper, zinc, etc., and emphasizes the significance of *occupations* attended by such intoxications. Charcot, in one of his later lectures, reported a case consecutive to cerebral rheumatism. Infection may explain some of the family groups of multiple sclerosis and Oppenheim's observation of the disease in a mother and child. From the numerous and often mixed infections noted, it seems unlikely that we have to deal with a specific organism. It is also a debated question whether the morbific microbes act through their elaborated poisons or by embolic colonization. It is, however, quite definitely settled that the irritative influence reaches the central nervous organs through the blood-channels and there

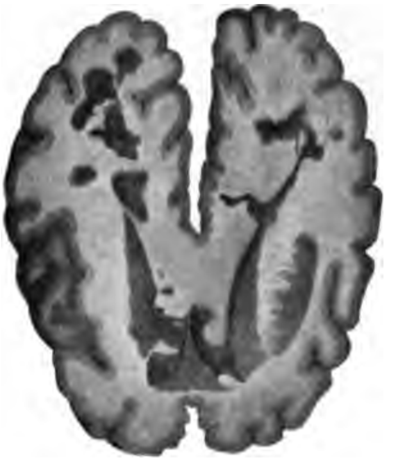

Fig. 204.—Lesions of insular sclerosis in the brain (Charcot).

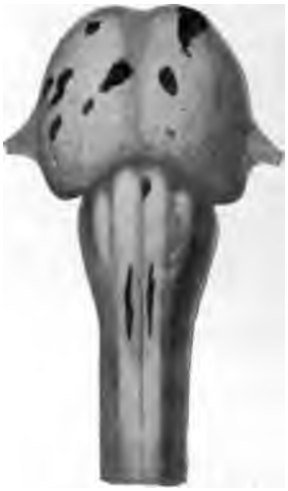

Fig. 205.—Lesions of the insular sclerosis in the pons and medulla (Charcot).

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[1] "Deut. Zeit. f. Nervenheilk.," Dec., 1901.

[2] Williamson, "Brain," 1896.

[3] "Berlin. klin. Wochens.," March, 1896.

sets up the localized sclerotic processes. Early sclerotic patches ordinarily show a centrally diseased blood-vessel, and thrombi have been found in several instances which Ribbert,[1] with Marie, is disposed to attribute to microbic emboli.

Goldscheider [2] declares the process analogous to that of an acute myelitis originating through the vascular supply, while Schuster and Bielschowsky [3] consider the change as primarily interstitial.

Morbid Anatomy.—*Macroscopically* the meninges retain their normal appearance. The pia mater is translucent and frequently the sclerotic patches on the cerebral and cord surfaces can be seen through it. These present various sizes and shapes and are usually grayish or pinkish gray in color. In some cases they may only appear after undergoing the action of bichromate and other hardening solutions. In distribution they recognize no law. Sometimes they are most frequent on the surface; sometimes they are confined to the interior of the cord and brain. In a general way they seem to prefer the white substance to the gray matter. They are found from the cerebral convolutions to the filum terminale and in variable numbers from a few to several hundred. Their dimensions are those of a millet-seed to a walnut or larger, and they are often of irregular, but almost invariably of definite, outline. They may invade the cranial nerves, especially the optic, and the spinal roots are not exempt. On section, they present a retracted appearance if old, a salient aspect if recent. Both sorts may be found in the same case.

Microscopically the definite outline of the plaques is still maintained, and they are sharply distinguished from the surrounding normal tissue. The *myelin* has disappeared from the nerve-fibers which pass through them, but the *axis-cylinders* usually persist and sometimes are enlarged. In recent plaques there is an abundance of *granular bodies* throughout the islet, but in older lesions they are confined to the periphery of the plaque. Through and everywhere in the plaque is a proliferation of the neuroglia, at once the evidence and the result of the irritant cause of the lesion. Observers are agreed that *secondary degenerations* do not, as a rule, follow the development of these sclerotic patches. Deprived of their myelin covering, insulation is probably impaired in

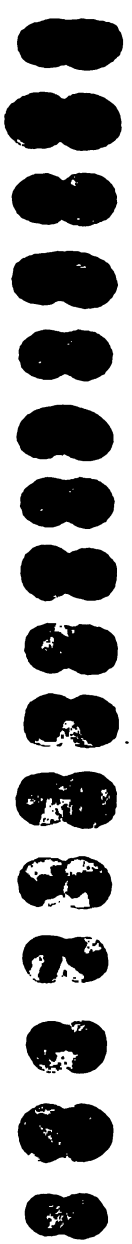

Fig. 206.—Distribution of lesions of multiple sclerosis in the cord. Sections taken at various levels throughout its entire length (Charcot).

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[1] Virchow's "Archiv," 1895.

[2] "Zeit. f. klin. Med.," Bd. xxx.

[3] "Neurolog. Centralbl.," 1897.

the nerve-fibers, and to this fact some writers attribute in part the trembling and other motor disturbances commonly presented. Occasionally myelinated fibers are found in old patches, and it has been suggested that they may be due to a regenerative process. These particularly occur in the pyramidal tracts and in Goll's columns. Popoff insisted that the alleged neuroglial hyperplasia does not exist, but regards this appearance as due to degeneration products of the myelin and axis-cylinders. Redlich,[1] however, in a later study, insists fully upon the glial increase in all cases. The nerve-cells embraced in the plaques are diminished in size, often pigmented, and their processes are atrophic or may have disappeared.

In the *optic nerve* there is the same interstitial increase without division of axis-cylinders, and, according to Uhthoff, papillitis and atrophy of the disc only occur when the plaque is situated immediately behind the globe.

The *vessels* in the plaques show decided changes. The coats are thickened, especially the external tunic, so that in section they remain open and dilated. The perivascular sheaths are also dilated and at times obliterated. In other instances thrombotic occlusion of the vessels has been observed. One vessel particularly altered is usually found near the center of each small sclerotic patch, and the sclerosis is most intense at and about this point. The appearance indicates that an early embolism or thrombosis sets up vascular lesions extending to the perivascular spaces, and entails irritative sclerosis of the adjacent neuroglia.

A certain number of cases must be kept in mind where the early changes are due to diffuse myelitis or cerebritis, or both, and in which secondary degenerations and numerous focalized softenings give rise to a similar clinical picture.

Symptoms.—Wide-spread as are the lesions and haphazard as is their location in multiple sclerosis, the clinical symptom group of the disease is tolerably uniform and ordinarily easily recognized.

The motor features constitute the most important group of symptoms in multiple sclerosis. The *gait* is usually disturbed at an early period of the disease. Sometimes it is clearly and *purely spastic*, identically the same as that presented in the paraplegic state: legs rigid, knees adducted, toes turned in and dragging along the ground; clonus showing in the trembling, shaking legs, which are pulled up and advanced by swaying movements of the body and pelvis; all reflexes exaggerated. To this is usually added more or less uncertainty of equilibrium, and we have a combination of cerebellar staggering and spastic rigidity which has been called the *cerebellospasmodic* gait, and is the one most frequently encountered. In exceptional cases the gait is purely *cerebellar* in quality, without any rigidity, and with diminished or normal reflexes. There is commonly more or less *muscular weakness*, which may reach a paraplegic degree and defeat all attempts at walking. In exceptional instances the lower extremities are not affected.

A considerable proportion of cases present *apoplectiform attacks*, followed by *transitory paralysis* at some period of the disease. In rare instances such an apoplectiform attack is the initial manifestation of the disease.[1] Sometimes both limbs and the face on the same side are involved; sometimes we have a crossed paralysis; sometimes the face escapes, and sometimes the palsy is paraplegic in distribution.

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[1] "Neurolog. Centralbl.," 1896, p. 562.

The upper limbs present not only the spastic weakness already noted in the lower extremities, but here is developed to its highest degree the so-called *intention tremor*, which is characteristic of the disease. While at rest the hands, trunk, and head are perfectly quiet; but once the patient attempts to reach any article, especially if the movement be somewhat extensive and requires precision, the entire member becomes animated with a coarse trembling which augments in amplitude as the movement continues. Finally the object is seized with considerable rudeness. The moment the arm is again placed at rest, and supported so that no muscular effort is expended, the tremor at once ceases. This tremor shows clearly in the handwriting of these patients, and writing may finally become impossible. Unlike the trembling of paralysis agitans, which is most pronounced in the fingers and while at rest, the intention tremor of multiple sclerosis usually arises at the roots of the extremities, and they are moved in their entirety from the shoulder- or hip-joints. This accounts for the *wide range* of the tremor excursions, and perhaps for their slow *rate*, which usually is between five and eight to the second. The lower extremities may also present this intention tremor to some degree, and it frequently shows in the *trunk* and *head* of the patient if without back-support or head-rest. The movements of the head are usually anteroposterior in direction, but may also be somewhat lateral and then tend to produce circumduction of the head upon the shoulders. Frequently the tremor is much more marked *on one side* than on the other, and rarely it is *unilateral*.

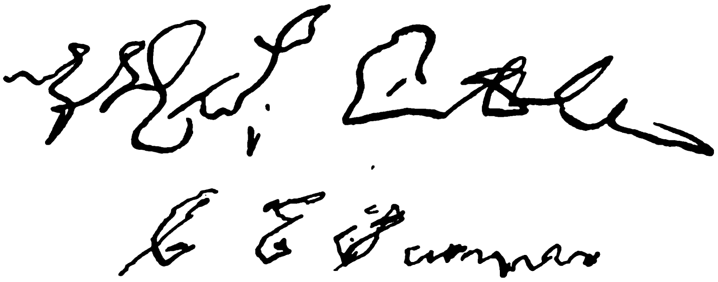

Fig. 207.—Handwriting in two cases of multiple sclerosis, showing irregular intention tremor.

One of the ordinary tests of this tremor is to direct the patient to take a drink from a tumbler of water. In marked cases the tremor appears directly the hand starts toward the glass. This is reached in a fairly direct line, grasped suddenly, and with increasing oscillations carried toward the lips, which are protruded to meet it, with the head and body advanced and shaking. The motions become more and more vigorous, and finally the glass is rattled or dashed against the teeth, and the water not already spilled may drench the patient's face and clothing.

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[1] Boulogne, "Rev. de Méd," May, 1893.

The little remaining is obtained by steadying one hand with the other, and firmly holding the glass with the mouth. This tremor is increased by *embarrassing emotions*, and is somewhat proportionate to the *extent* and *duration* of the movement attempted.

Sensory features in multiple sclerosis, in a general fashion, may be said to be insignificant, so far as *general sensibility* is concerned. Occasionally a patient complains of anomalous sensations of heat or cold, formication, fullness in the limbs, and sometimes of pains which may even be lightning-like in character or of the girdling form. According to Freund, the slight objective sensory changes sometimes encountered are confined to the fingers and toes mainly, and to the distal portions of the limbs. They consist of disturbance of touch, of hyperalgesia and analgesia, and insignificant modifications of thermic sensibility. They are often transient and changeable. F. von Gebhardt[1] claims that in advanced cases certain areas present permanently disordered sensation. Hemianesthesia and other hysterical distributions of anesthesia sometimes complicate multiple sclerosis, as the diseases may be, and often are, associated.

Hearing, *taste*, and *smell* are singly disordered in rare cases of multiple sclerosis, usually in the direction of deficit, and may be completely lost.

Disturbance of *sight*, on the other hand, is frequent, often very marked, and of great diagnostic value. The ocular apparatus may be affected in any portion of its neuromuscular make-up. *Nystagmus*, usually consisting of lateral movements, is presented by over half the cases, perhaps in seventy per cent. True nystagmus, in which the eyes constantly oscillate, occurs about once out of five such cases, the other four presenting nystagmiform movements only on occasion, particularly when the eyes are forcibly deviated to the right or left. It should be sought by directing the patient's line of sight throughout the entire lateral and vertical range of ocular movement. In seventeen per cent. of cases Uhthoff found *paretic* conditions of the extrinsic eye-muscles. These, like the hemiplegic features, are usually transient. They may constitute a complete *external ophthalmoplegia*, or be confined to any of the motor oculi nerves, or to portions of the third cranial pair. They are, as a rule, *bilateral* and affect the *associated movements*, showing their nuclear nature. The nystagmus is of a similar nature, and probably due to the disturbed muscular balance.

The *pupils* may show miosis, inequality, and diminished reflexes to both light and accommodation, but the Robertson pupil is practically unknown, and miosis is usually associated with an exaggeration of the light reflex.

The *optic nerve* is very frequently affected and vision is often modified. The papilla may show *optic neuritis* or *atrophy*, but the atrophy is very seldom extreme, and the disc usually retains some color in a part of its expanse. Some papillary modification is found in over one-half of all cases, and some cases showing no changes in the nerve-head still present visual defects. *Vision* may be slowly or rapidly reduced and blindness may follow, but the amblyopia is ordinarily transitory, and a considerable degree of sight is frequently restored. Central scotomata, irregular or regular retraction of the field, dyschromatopsia, particularly for red and green, may be variously combined in the field, and in these respects the ocular disturbances are rarely symmetrical and may be entirely unilateral. They may be of insidious or rapid development and may be early or late manifestations of the disease. They bear a rough relation to the intensity of other symptoms and often increase correspondingly with them.

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[1] "Deut. Arch. f. klin. Med.," Bd. lxviii, No. 1 u. 2.

Visceral disturbances are insignificant in insular sclerosis. Rectal and vesical incontinence or retention, gastric crises, and other similar manifestations of organic disease of the cord are sometimes encountered, but are not severe in degree or constant in appearance.

Trophic disturbances are unusual. Loss of nails, gluteal decubitus, and localized wasting of muscles, particularly of the interosseous muscles of the hand, have been met with.

Bulbar Symptoms.—Difficulty in swallowing and in mastication, trembling of the tongue, inability to protrude it, glycosuria, and polyuria are occasionally encountered, and, with the other bulbar symptoms already noted, point to the location of sclerotic patches in that region.

Cerebral Features.—Among the most characteristic symptoms of multiple sclerosis is the *speech defect.* This is very commonly present and consists of a *slow, monotonous, scanning* pronunciation that slights no syllable and ends abruptly, *spasmodically,* explosively with the last. It sometimes causes elision of the final sounds or every syllable may be slurred. There is a distinct effort to articulate each sound with rests between. This staccato speech, in marked cases, is well marked, but in some instances it may lack some of its features or entirely default. Its mechanism is of difficult explanation, but is, at least in some cases, attributable to faulty action in the articulative muscles.

Vertigo is sometimes present and may disturb the patient at rest or in walking. In some cases the nystagmus causes objects to dance before the eyes; in others, with ocular-muscle paresis, there is diplopia, both of which may cause vertigo. In rare instances the vertigo presents Ménière's type. The *apoplectiform* and *epileptiform attacks* that have been already incidentally mentioned, *spasmodic laughter* and *crying* and slight *mental enfeeblement,* with *indifference* and *dejection,* indicate cerebral lesions. In rare cases an expansive, egotistic delirium of grandeur may appear closely resembling that of paretic dementia, and complete dementia may ensue. Hunt[1] insists that paretic dementia and multiple sclerosis are sometimes combined in the same subject.

Hoffman,[2] in the 100 cases on which he based his study, found speech difficulties in 54, giddiness in 64, headache in 40, optic atrophy in 50, nystagmus in 56, intention tremor in 71, paresthesiæ in 66, and objectively disturbed sensation in 28. The sexes were about equally represented by 47 women, 53 men.

Course and Forms.—The *onset* may occur abruptly by an apoplectiform attack, followed immediately by hemiplegic or paraplegic or monoplegic weakness, or these may suddenly appear without the convulsive attack. In other instances vertigo or visual disturbance suddenly opens the program. More frequently the onset is gradual and the course progressive. Difficulty in walking or in speaking or the trembling is first noticed and gradually increases, leading in the other prominent features of the malady. Sometimes the initial feature consists of attacks of pain of a stabbing, lancinating variety. The advance of the disease may be chronically progressive, or it may show exacerbations or remissions, and continue finally to advance. Sometimes the amelioration is permanent and recovery is possible. In progressive cases death may result from intercurrent maladies, from an apoplectiform attack, from bulbar accidents, or from the exhaustion of terminal dementia.

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[1] "Am. Jour. Med. Sci.," Dec., 1903.

[2] "Deut. Arch. f. klin. Med.," Bd. lxviii, No. 1 u. 2.

The duration of the disease, like its anatomical basis and its symptoms, is very variable. It may reach full development within a few months or a year, rarely almost at a bound. It may terminate in a year, last twenty, or recede completely at any time.

In some instances only one or a few of the most notable symptoms of the disease ever develop. These cases constitute the *formes frustes* of Charcot. Thus the trembling, or the speech defect, or the gait with or without nystagmus, or bulbar symptoms and amyotrophy may predominate, while other symptoms are only very slightly developed.

Diagnosis.—In a well-developed case the diagnosis may be made. The intention tremor, cerebellospasmodic gait, spastic weakness, nystagmus, and syllabic speech make a positive picture. The differentiation from *family ataxia* may, however, present great difficulty when several members of the same family are affected with multiple sclerosis. Friedreich's disease is marked by flaccidity and abolished knee-jerks, the motor difficulty is purely ataxic and never spasmodic, and optic-nerve symptoms are practically always absent. In the heredocerebellar type of family ataxia there are increased reflexes and often optic atrophy, but a long family succession of cases rarely fails us, and the motor difficulty here again is devoid of spasticity and is purely ataxic. In neither type do we find the well-developed syllabic speech nor the intention tremor, though the speech is modified and ataxic and there are choreic movements which might at first mislead. Cases clinically mistaken for *amyotrophic lateral sclerosis* have been recently reported by Probst and Brauer, one for *transverse myelitis* by Siemerling, and one for a *Brown-Séquard paralysis* by Jeremias.

Hysteria is capable not only of mimicking every objective symptom of insular sclerosis, but sometimes is associated with it in the same patient. It should never be out of mind in making the diagnosis, and the stigmata of the neurosis should be faithfully sought in every instance. Cases showing hysterical signs and the symptoms of multiple sclerosis, according to Buzzard,[1] should be looked upon as probably organic if the tendon reflexes are exaggerated and, at the same time, the plantar reflex is abolished. This observer has noted optic atrophy in one-half of his cases of insular sclerosis, and found Babinski's toe-sign in all in which it has been sought.

In the partial cases, mainly marked by one of the prominent char-

[1] "Brit. Med. Jour.," May 6, 1899.

acteristics of multiple sclerosis, the diagnosis is decidedly difficult. In every such instance all the features of insular sclerosis should be sought, and some of them, if only partially developed, will be found to fix the diagnosis, or the presence of signs of other diseases will guide. When trembling predominates, we must exclude paralysis agitans, metallic intoxications, chorea, and hysteria. When the speech defect is most prominent, we must think of paretic dementia and the family ataxias. In cases showing a predominance of cerebellar ataxia intracranial tumors and the family ataxias must be excluded. When the gait is purely spastic, we must exclude transverse myelitis and the combined scleroses of the cord.

Prognosis.—While the outlook in a case of multiple sclerosis is always grave, the tendency to remissions and the occasional complete recession of the disease make it distinctly more hopeful than in the destructive sclerotic lesions of tabes and cross-myelitis. It is impossible in a given case to forecast its probable course so long as it is progressing. Apoplectiform seizures followed by paralytic features and aggravation of all the symptoms may occur at any moment; but when the disease has shown remission and improvement we are justified in hoping, with due reservation, for better things.

Treatment.—In every case the nature of the origin of the malady will guide therapeutics. If the disease is to be considered as mainly due to infections and to the continuous activity of microbic life, which from time to time leads to new plaques or increases old foci, bactericidal preparations are indicated—quinin in malaria; mercury, iron, arsenic, and salicyl preparations in other septic conditions. Generally speaking, an antisepticemic régime is in order. This may only be valuable when the general resistive powers of the economy are increased by the best hygienic and general health measures. The future may furnish some organic antitoxin more powerful than our present medicaments.

CHAPTER II.

SYPHILIS OF THE NERVOUS SYSTEM.

THE syphilitic lesions of the nervous system, especially of the central apparatus, are of extreme frequency and of almost infinite variety. In this section little more can be attempted than an enumeration of them, with special reference to clinical peculiarities and the most efficacious mode of treatment.

Syphilitic nervous disease may be divided into (1) those due to the active ravages of the virus and (2) those which follow the infection and are not marked by neoplastic products. The one is *specific*, the other *parasyphilitic.* The first is microbic, the second toxic or chemical. Such a view of luetic lesions and consequences gives us the best working basis at the present time. Syphilis is capable of transmission to offspring in either form, and we have some hereditary syphilitic diseases of the general nervous system marked by the ordinary gummatous processes, and others due solely to faults of nutrition and toxic action. Again, both the syphilitic and parasyphilitic lesions may be present in the same individual at the same time or at different periods. The following tabulation may serve to systematize the subject:

<table>
<tr><td rowspan="5">Syphilis of the General Nervous System</td><td rowspan="3">Active specific lesions</td><td rowspan="2">Cerebrospinal syphilis, acquired and hereditary (early and tardy)</td><td>Cerebral</td><td>Meningitis
Cerebritis
Arteritis
Neuritis</td><td>Mania
Melancholia
Pseudoparesis</td></tr>
<tr><td>Spinal</td><td colspan="2">Meningomyelitis
Myelitis
Spinal paraplegias</td></tr>
<tr><td colspan="4">Syphilis of nerves.</td></tr>
<tr><td rowspan="2">Parasyphilitic diseases</td><td>Acquired</td><td colspan="3">Tabes
Paretic dementia
Optic atrophy
Neurasthenia
Hysteria
Epilepsy
Neuralgia</td></tr>
<tr><td>Hereditary</td><td colspan="3">Juvenile tabes and paresis
Infantilism
Hydrocephalus
Cerebral or spinal agenesis</td></tr>
</table>

Specific lesions of the general nervous system, like specific lesions elsewhere, originate in the connective tissue or blood-vessels and secondarily affect the parenchyma, disturbing function first and finally destroying the essential cells and fibers of the part. Rarely they arise in bony structures and thence invade the adjoining nervous apparatus or act upon it by pressure. Their peculiarity is in the gummy plastic exudate and its formative tendency. It is essentially a neoplastic process, which may vary from an appearance of simple inflammation to the accumulation of sizeable tumors. In early stages there is often a marked tendency to regressions and recidives. Old lesions, if of considerable size, may caseate within and cicatrize upon the surface by fibrous organization and fibroid changes may alone remain to indicate the preëxisting gummy condition.

Acquired syphilis affects the central nervous system by specific lesions in a very considerable proportion of all luetic cases. Hjelmann, leaving out of the question both tabes and paretic dementia, states that from 1½ to 2½ per cent. of all cases of syphilis develop cerebrospinal lesions, and that of those presenting the tertiary features of the infection, twelve per cent. show invasion of the cerebrospinal axis. Fournier puts this latter percentage at twenty-one. In the great majority of cases the syphilitic process is wide-spread and somewhat haphazard in its location. Very rarely do cases of spinal syphilis lack evidences of cerebral involvement at some period, but cerebral syphilis, on the contrary, is sometimes devoid of spinal complications. We may, for descriptive purposes, divide these cases of acquired active syphilitic lesions into the cerebral and the spinal. Their common association should never be forgotten.

CEREBRAL SYPHILIS.

Syphilitic cerebral meningitis is perhaps the most frequent form of cerebral syphilis, and usually, to some extent, complicates all other varieties of encephalic lues. The *favorite location* is the basilar area, especially the interpeduncular space, from which it frequently extends upward on to the convexity of the parietal, and particularly of the frontal, lobes. So commonly is the base affected that, speaking clinically and according to Charcot, it may never be considered exempt, even when the symptoms point to the convexity alone. The distribution recalls that of tuberculosis. A gelatinous transparent substance infiltrates the soft meninges about the circle of Willis and the optic chiasm. It is composed of small, round, embryonic cells, and is very vascular and viscoid. Caseation and sclerotic degeneration mark the lesion later. The adjoining or enveloped nerves and vessels may be injured. Neuritic atrophy on the one hand and obliterating arteritis on the other are thus set up. Extension of the process to the nerve-sheaths and interstitial structure accounts for the very frequent symptoms on the part of the cranial nerves in cerebral syphilis.

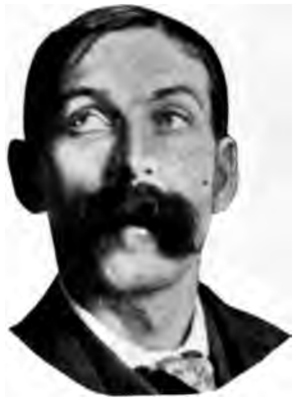

Fig. 208.—Ocular palsy in a case of brain syphilis in which at one time there was a crossed hemiplegia, right face and left extremities. The right eye overacts, turning upward and outward in looking to the right.

In the field of syphilitic meningitis gummy masses, or *gummata*, are usually encountered. They vary in size from miliary bodies to tumors as large as filberts, which may be solitary, few, or many in number. Their favorite location is at the base or on the convexity, especially on the frontal lobes, but no portion of the brain is exempt. Sometimes they form on the surface or in the thickness of the dura mater, or deeply within the brain, on some septal infolding or penetrating blood-vessel. They comport themselves as do brain-tumors generally, and give rise to similar localizing signs and symptoms. Sclerous or fibrous degeneration marks the later stages of all these specific neoplastic lesions, and gives rise to what is sometimes called sclerous syphilitic meningitis.

Syphilitic cerebritis may exist as a direct extension from a specific meningitis or gumma, or may occur more or less independently. It may present itself as a diffuse, gummatous encephalitis, or as localized encephalic gummata which tend to caseate, or it may appear in circumscribed patches or plaques, especially in the pontine and peduncular surfaces, which may end in sclerous patches. Every syphilitic process in the brain entails a surrounding zone of cerebral softening or cerebritis.

Syphilitic Cerebral Arteritis.—Syphilitic endarteritis in the brain is a comparatively common accident. It may result in the obliteration of the large basilar vessels secondarily to a gummatous meningitis, or it may develop as a primary focus of syphilitic activity. Its legitimate result is a localized ischemia, which, if complete, results in thrombotic brain-softening. Heubner insists upon its frequency in the basal ganglia. The middle cerebral arteries are frequently affected either in the numerous small branches of the Sylvian or by the complete obliteration of this motor-zone vessel.

It is also established that cerebral hemorrhages may follow a specific arteritis. This rarely occurs within the brain-substance, but more often takes place in the large and comparatively unsupported basilar vessels, resulting in a foudroyant apoplectic stroke almost immediately fatal.

Syphilitic Lesions of the Cranial Nerves.—Owing to their exposed position at the base, where specific cerebral invasion is most common, the cranial nerves are very commonly implicated. The relative frequency with which they are affected diminishes from the chiasm and optic nerves backward toward the bulb. The order is as follows: Optic nerves, motor oculi, abducens, trifacial, facial, auditory, and only rarely the glossopharyngeal, vagus, and hypoglossus.

The motor oculi, or *third nerve*, is affected in about forty per cent. of the cases, and shows a particular vulnerability on the part of the fibers supplying the levator of the upper lid. Hence the frequency and significance of ptosis. Inequalities of the pupil and pupillary stasis are of equal importance and frequency. A rigid pupil should always raise a suspicion of syphilis. It is to brain-syphilis what the Robertson pupil is to tabes. Referable to the affection of the chiasm and optic nerves and tracts we find all forms of irregular and unequal contractions of the visual fields. Optic neuritis is very frequently present and atrophy may result. The trifacial may be involved in one or all its branches, causing disturbed sensibility sometimes, but more frequently neuralgic pains of corresponding distribution. Infrequently facial palsy is encountered, and in rare cases this is associated with deafness on the same side.

The amount of injury to a nerve varies within wide limits, but often presents the suggestive peculiarity of advance and recession, giving rise to temporary, *fugaceous palsies* and *temporary sensory features*, such as amblyopia, lasting a day or two. After several such attacks the disability is likely to persist permanently and indicates a destructive change in the nerve, which commences as an interstitial infiltration and neuritis.

The general symptoms of cerebral syphilis are modified by the particular lesions that are present. These, as a rule, are *multiform*, but with special prominence of certain features. One case attracts most attention by its eye symptoms, another by its neuralgic pains, another by its convulsive manifestations, another by its stupor or mania or paralysis.

There is always, or nearly always, an initial period—a *premonitory period*—that it is of the greatest importance to recognize. Then only does medication offer a fair chance of effecting a cure. Once passed, irreparable damage is done, and the best that can be hoped is to check further mischief and leave the brain and nerves scarred with cicatricial tissue. Brain-syphilis appears, ordinarily, during the secondary period, but may occur at any date after the first few months. Thus the first year shows the greatest number of cases, which diminish gradually, year by year, to the end of life. There is no period of exemption after infection. Cases of brain-syphilis are encountered twenty, thirty, and forty years after the initial sore, and probably many syphilized individuals die from other causes and thereby escape luetic brain accidents. If tertiary manifestations appear, the likelihood of the occurrence of brain-syphilis is increased. It is important to know that a blow or jar to the skull may precipitate and focalize syphilis in the brain, and may greatly aggravate it when present. The same is true of mental shock and fright. The phenomena of the premonitory stage consist of *headaches, disturbances of sleep, mental changes,* and *physical decline.*

The *headache* in its typical form is atrocious in severity, often circumscribed, and deeply seated. It is frequently marked by local tenderness on percussion and shows daily, usually nocturnal, exacerbations. The syphilitic headache may remit without apparent cause, or an exacerbation may be followed by a ptosis or an amblyopia, or it may terminate upon the appearance of a hemiplegia or other serious secondary brain-lesion. Usually the only sedative capable of controlling it is morphin, but it yields in a few days to either mercury or the iodids. While the headache is mainly vesperal or nocturnal, in some instances it is most severe in the morning and some trace of it usually lasts throughout the twenty-four hours. Commencing in a localized area, it may gradually invade the entire head, and frequently, if unchecked by proper treatment, lasts weeks and months.

The *sleep* is modified in two ways. There is *insomnia* or *sopor.* The nocturnal headache in itself may destroy sleep at night, but in addition the patient often fails to sleep after the headache subsides. *Insomnia* often occurs early, before the headache has grown intense. Later, there is usually a tendency to *stupor,* and this also may appear early. The patient drops to sleep at any time during the day, at his meals, over his paper or desk. Aroused at night by the cephalalgia, he drops back into a heavy, stuporous .sleep, and often requires vigorous rousing. In a somnolent way he may get up for any purpose and at once drop to sleep on returning to bed. The stupor may become continuous and reach a comatose depth.

Mental changes ordinarily accompany the cephalalgia. The patient's wits are dulled. He is apathetic, disinclined for study, work, or social enjoyment. The memory is frequently impaired. He looks and acts and feels depressed and indifferent. His ideas come slowly and he may show irritability or become greatly depressed.

The general health often fails. The appetite is commonly lost early, strength diminishes, flesh is lost, and the skin and mucous membranes grow pale and anemic. Often at this time vertigos, tingling in an extremity, inequalities in the pupils, a drooping lid or some twitching of a limb indicate the impending storm and show its direction.

Special Symptoms.—After the premonitory period or even from the first the disease shows one of three major tendencies, marked by the preponderance of *meningitis, arterial accidents,* or *tumor formation,* respectively. These may be and usually are variously combined.

The meningeal form is marked by extreme depression and torpor. The patient lies like one narcotized or stupefied with alcohol. At times he can be roused to answer in monosyllables, but promptly subsides into the lethargic state and may become unconscious and comatose for a few moments, for an hour, or for several days. When he talks it is in an uncertain and unreliable way. Frequently there is a little delirium. His headache alone seems to cause him trouble. Usually pupillary sluggishness and inequality are present, ptosis and squints are frequent, and diplopia sometimes admitted. There may be urinary incontinence or retention. Fever is ordinarily, but not invariably, absent and the physical functions are well carried on. Death rarely results from this form of cerebral syphilis, but, on the other hand, complete recovery is equally rare. Some trace in the way of mental apathy is likely to persist. In exceptional instances there is wild, maniacal delirium and motor excitement, an elevated temperature, and quickened pulse.

The Arterial Form.—Cerebral syphilitic arteritis is usually accompanied by basilar specific meningitis and the premonitory headache. Cranial-nerve symptoms are also usually present. It gives rise to thrombus and cerebral softening, resulting in hemiplegia or monoplegia, or aphasia or other localized cortical disability. Usually this does not come on at once, but by slight premonitory strokes and numerous "warnings." Transitory plegias and aphasias are always suggestive of syphilis. When the motor cortex is involved, spasm or partial epilepsies are commonly induced. The rule applies here, as in gummata, that epileptic seizures after the age of thirty are generally due to syphilis, and that paralytic seizures in adults, before the age of forty, in

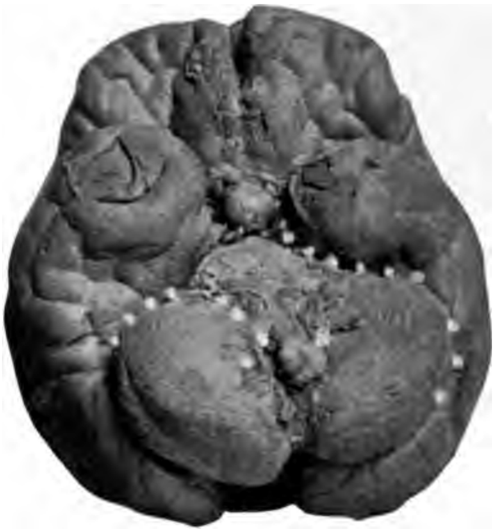

Fig. 209.—Brain showing basilar syphilitic meningitis, with generalized exudate and small cysts in the deeper furrows (Dr. W. A. Jones).

the absence of cardiac and renal lesions, are almost invariably syphilitic. Another indication of syphilis is a multiplicity of lesions. A double hemiplegia or left hemiplegia with aphasia or pseudobulbar palsy from double-sided lesions, or cerebral hemiplegia and paraplegia from brain- and cord-lesions, in the same case, speak for syphilis.

Hemorrhage, as already indicated, is usually from a large basal vessel and is promptly fatal. It is commonly secondary to aneurysmal degeneration and is a rare cause of death in syphilitics. Occasionally thrombosis, beginning in the circle of Willis, eventually reaches and obstructs the basilar artery, and death follows coma.

The gummatous form of cerebral syphilis is usually marked by focal symptoms, and here all the rules of cerebral localization apply. As above indicated, the favorite location for gummata is at the base and upon the frontoparietal convexity, precisely the regions in which focal symptoms arise. These consist of cortical states of irritation and destruction,—in other words, of limited epileptic attacks and cortical palsies, and of cranial-nerve lesions. Usually the focal signs and symptoms in gummatous cases are preceded by the premonitory phenomena. This might be expected, as the usual meningeal or cortical gumma is secondary in point of time to a more or less diffuse specific meningitis. In rare cases the convulsion is the first manifestation of cerebral invasion. Generalized fits and epileptiform convulsions are not rare in diffuse cortical syphilis, and may be associated with the Jacksonian attacks, petit mal, momentary aphasias, sudden "thickening of the tongue," and similar evidence of circumscribed lesions. Syphilis also seems capable, according to Fournier, of producing a parasyphilitic epilepsy to which attention will be recalled. (For further details regarding cerebral gummata the reader is referred to the chapter on Cerebral Tumors, page 255.)

Syphilitic Mental Diseases.—The mental disturbances of brain syphilis are those of organic brain disease, but in subjects strongly predisposed to insanity the syphilitic cachexia may induce ordinary forms of insanity. Morbid mental states due to the encephalic ravages of lues are most frequently of the depressed varieties grouped under the generic name of melancholia, with a decided tendency to dementia. Less frequently there is exaltation and delirium, which may be intense, and in both forms hallucinations are common. Signs of gross brain-lesions are rarely wanting. Cranial-nerve symptoms, localizing fits, monoplegic palsies, and speech defects point the way to diagnosis.

In some instances symptoms arise that closely ape those of paretic dementia, and give rise to the term *syphilitic pseudoparesis.* The differential diagnosis may, indeed, be impossible. The features suggesting active syphilitic disease, as contrasted with the degenerative changes of paresis, are a less steadily progressive course, less expansiveness, less amnesia, less speech difficulty, absence of the Robertson pupil, presence of cranial-nerve palsies and early monoplegias, and improvement under antisyphilitic treatment. Syphilophobia and hypochondriasis, arising from the knowledge or fear of being infected, have only an indirect relation to the disease.

Diagnosis.—The diagnosis of encephalic syphilis, when there is a plain history and marked evidences of previous infection, ordinarily presents no difficulties. In cases where such data are lacking, one of the nicest of diagnostic problems is furnished. *Nocturnal headaches* of the character described, *epileptoid attacks* occurring after the age of thirty, or *apoplectic attacks* appearing before the age of fifty, should at once, singly or in combination, raise the suspicion of syphilis. A history of any sort of a genital sore strengthens the case; and if, in addition, the slightest secondary manifestation can be detected or recalled by intelligent cross-questioning, small doubt remains. It may be stated that the great majority of cases of syphilis of the nervous system present very slight secondaries, or these may have entirely escaped attention. Another indication of syphilis in brain disease is a *multiplicity of lesions*. Cord and brain symptoms in the same patient are significant, as are double-sided brain-lesions or lesions of both base and convexity. The almost constant presence of basilar gummy meningitis, with its consequent *cranial-nerve symptoms*, among which are ptosis, pupillary abnormalities, and optic-nerve disturbances, often alone is sufficient to guide the diagnosis. The *mode of development* counts for something. In brain-syphilis the onset is rarely sudden, but is, rather, by repeated advances, with remissions. *Fugaceous palsies* and fleeting sensory losses are the rule in syphilis. The phenomena of the *premonitory phase* can not be too strongly insisted upon. *Sterility* in male or female should be given a certain weight, and repeated miscarriages frequently incriminate the husband. Finally, the *therapeutic test* stands for considerable. If under adequate alterative dosage the disease yields within a week, it sustains the diagnosis to some degree; but we must never forget that in other organic processes, even in sarcomatous tumors, the iodids may cause a notable remission in the symptoms.

Prognosis.—The outlook is far from favorable, though it is too commonly assumed to be so solely because syphilis is at the bottom of the cerebral symptoms. It may be safely stated that less than one-half of the cases of brain-syphilis completely recover. Fournier, in 90 cases, reported 23 unrelieved, 13 improved, 14 died, and 30 recovered. Of those recovered, probably many presented recurrences later in life. In a general way we may expect to check the progress of active syphilitic processes excepting in rare cases of a malignant and unmanageable character, where the human organism can not tolerate a medication intensive enough to permanently subdue the disease. The golden opportunity is in the prodromal stage. Lesions that have caused destruction of nervous tissue, such as results from thrombotic softening, secondary degeneration, hemorrhagic disintegration, and sclerotic strangulation, leave indelible and irremediable effects. In view also of the known ability of syphilis to lie latent and dormant for years, and its clinical remissive tendency, the wise physician will always maintain great reservation in even the most promising cases. Marked and prolonged stupor and decided mental disturbance make the expectation of complete restoration very slight. A patient who has once developed syphilis of the cerebrospinal apparatus is never safe and must maintain medical supervision, and should undergo courses of treatment at yearly intervals as long as life lasts.

Treatment.—The treatment is the same as that in the spinal form, now to be considered, and will be detailed in that connection (page 482).

SPINAL SYPHILIS.

Nearly if not every case of spinal syphilis sooner or later presents evidences of cerebral invasion, and the diagnosis of syphilis of the cord often depends upon this association. It is proposed in this section to mainly emphasize the cord-lesions.

Syphilis of the spinal cord and membranes is somewhat more precocious in its appearance than brain-syphilis, and most frequently appears from the third to the sixth month of the syphilitic history. It then diminishes in frequency during the rest of life. As within the skull, spinal syphilis is a disease of the meninges, nerve-roots, and blood-vessels. The cord-changes are invariably secondary and, in a sense, mechanical. It may involve any or all of the membranes as a specific meningitis, which sometimes is marked by gummatous thickening and tumor formation, but much less commonly than is the case within the skull. The nerve-roots and pial vessels are frequently involved in the specific meningitis, which may also extend to the periphery of the cord. Another group of conditions arises from impairment of the blood-vessels. Endarteritis, endophlebitis, and thrombosis may cause disturbance of the circulation in any part of the cord. They may cause softening and changes that are clinically similar to a focal myelitis, or, by affecting the blood-supply of the posterior arterial field, set up the changes in the posterolateral portion of the cord that induce combined scleroses. Ordinarily, spinal syphilis is multiform, and a varied picture is presented through the implication of various parts of the cordal apparatus. The matter is still further complicated by the variations in a single case, which may at different times present great modifications. Thus a case beginning as meningitis may become a meningomyelitis, set up a combined sclerosis, and terminate by focal softening, consuming many years in its history. For descriptive purposes, and based upon clinical manifestations, we may divide cases of spinal syphilis into: (1) Meningitis and meningomyelitis, (2) acute myelitis, and (3) ataxic paraplegias or combined scleroses.

Syphilitic Meningitis and Meningomyelitis.—These furnish the most habitual expressions of spinal syphilis. In clinical features they differ but little from similar lesions arising from any other source. The principal symptoms are pains and palsies. The *pains* are onset features and are of extreme intensity, usually with a well-marked tendency to nocturnal exacerbation. They are usually located in the lower extremities, but with these there is generally associated a most intolerable *rachialgia* in the lower part of the back. They are *root pains* and may be marked by tenderness along the nerve-trunks, and even by herpes and glossy skin. Partial palsy of certain muscle groups, with disturbed sensation in the cutaneous area of identical innervation, is occasionally seen, and still further confirms the neuritic and root disturbance.

The *palsies* are rarely complete. There is a feeling of weakness in the legs, sometimes distinctly greater on one side than on the other; a paraplegic distribution that may ascend to any level, but most frequently remains below the waist-line. In rare cases the upper members are most or alone affected. In some cases a pachymeningitis cervicalis has been found, with its characteristic deformities, and in some such instances the spinal portion of the dural disease has been a downward extension from syphilitic thickening within the skull.

The cord is frequently affected, as is shown by bedsores, sphincter disturbances, Brown-Séquard paralysis, and a tendency to spasticity in the lower extremities, which are at first relaxed and enfeebled.

The sensory disorders usually open the program and the paraplegia is ushered in by a feeling of heaviness and clumsiness in the legs. Usually there are a series of remissions and advances until the spasmodic paraplegia is finally established.

Occasionally, gummata form in the meninges and give rise to tumor symptoms or the Brown-Séquard syndrome. Multiple miliary gummata are still rarer.

In the annular invasion of the cord the blood-supply is interfered with, both arteries and veins suffering from deformity and endothelial changes that may lead to ischemic softening and hemorrhage. Combined scleroses are similarly induced.

Syphilitic acute myelitis, or softening, is not a rare condition. The best of recent observations indicate that the origin is in thrombotic occlusion of blood-vessels, syphilis acting as any other infection, or by setting up a specific arteritis or phlebitis, with or without accompanying meningitis. In the softened area there is commonly great syphilitic cellular activity, and secondary hemorrhage frequently causes a hematomyelic condition. The favorite location for softening is in the gray substance, and the thrombosis may extend to all the vessels of a considerable area. Very rarely does the lesion involve the entire cross-section. Secondary ascending and descending degenerations naturally follow.

The symptoms are those of an acute myelitis: sudden onset, paraplegia, corresponding sensory disturbances, sphincteric incontinence, and spasticity after a few weeks.

Syphilitic Ataxic Paraplegia.—This variety of spinal syphilis has been much written about since Erb, in 1892, called attention to its frequency and uniformity. He proposed for it the name of *syphilitic spinal paralysis.* Subsequent communications by Muchin, Kuh, Kowalewski, and others tended to give it an autonomy that Erb specifically deprecated in his earlier reports. Recently [1] he asserts for it the dignity of a distinct clinical type due to primary changes in the lateral tracts of the cord following syphilis in the same sense that posterior sclerosis is post-syphilitic. Oppenheim, Brissaud, Marie, and others have denied its entity.

The condition is usually of insidious onset, developing in a few weeks, months, or years. The first symptoms are feelings of heaviness and paresthesia in the legs, very soon accompanied by difficulty in emptying the bladder through spasmodic action of sphincter and detrusor. Rigidity and spasticity in the legs soon develop, and the gait shows them in the dragging toes, adducted knees, and uncertain steps. Knee-jerks, rectus and ankle-clonus, and the toe-sign are commonly well marked and muscular strength is more or less reduced. Spasticity and a lack of muscular strength are especially insisted upon by Erb. Sensory disturbances are slight or entirely absent, and the disease is very slowly progressive or inclined to be stationary or to even improve under treatment for syphilis. It seldom reaches a stage of complete helplessness. Often the upper extremities are affected in a less degree, and pupillary symptoms are not uncommon.

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[1] "Br. Med. Jour.," Oct. 11, 1902.

In this outline it is easy to see the symptoms of posterolateral sclerosis or ataxic paraplegia, or the combined scleroses (see p. 448). The condition may also arise secondarily from a meningomyelitis, and it is clearly due to vascular disturbance in the posterior arterial cord-field, with the ataxia, spasticity, and paraplegia that mark such lesions.

Diagnosis.—The diagnosis of spinal syphilis is frequently of the greatest difficulty. Unlike cerebral syphilis, it has not a characteristic premonitory stage. The nocturnal rachialgia and the advance and retreat of the spinal symptoms have some significance, but a history or the evidence of syphilitic infection has more weight and the presence of encephalic disease has most of all. This last, as already frequently stated, rarely defaults. In the combined scleroses we must exclude tabes, which can usually be done by a tap on the knee and an examination for objective signs.

Prognosis.—The outlook in focal myelitis is the same as in that lesion from other causes than syphilis, and depends upon the amount of damage to the cross-section, the vertical location, and the secondary degenerations. Meningomyelitis can frequently be held in check and offers a good prognosis if the cord is only superficially injured. The prognosis of the combined scleroses is good as to life, but bad as to complete recovery.

Syphilitic neuritis is not a common accident. In rare instances the toxic condition may induce a multiple neuritis. Cestan [1] was only able to collect 14 cases from the literature, but in minor degree it is probably not infrequent. Usually single nerves, as the sciatic, intercostal, or some branch of the brachial plexus, are affected, and such lesions are often bilateral.

Hereditary Cerebrospinal Syphilis.—Hereditary syphilis is capable of provoking any and all of the injuries of the general nervous system that follow the acquired infection, and, like it, is often marked by a multiplicity of lesions in a given case. These may appear congenitally, in the early years of life, or be tardy and postponed until puberty, or even to the twentieth and thirtieth year. The presence or history of other syphilitic stigmata, such as Hutchinson's teeth, hydrocephalus, idiocy, cachexia, syphilitic skin diseases, choroiditis, and a history of parental syphilis, must often be relied upon for a diagnosis. The lesions of hereditary syphilis are less amenable to treatment and of worse prognosis than those of the acquired form.

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[1] "Nouv. Icon. de la Salpêt.," 1900.

Treatment of Active Cerebrospinal Syphilis.—The moment syphilis of the cerebrospinal apparatus is discovered, or even strongly suspected, intensive treatment should be instituted. The diagnosis of brain-syphilis implies the recognition of a critical condition. A number of details are important to secure toleration and to maintain the prolonged use of specifics. If the case be urgent,—and in view of the location of the disease it invariably is,—both iodids and mercury should be used at once. If the lesion is arterial, preference should be given the iodid of sodium, as the potassium salts have a tendency to increase arterial tension, and the sodium is also usually better tolerated. The writer's plan is to commence with 20 grains three times a day, and increase the dose 10 grains daily until, in rebellious or critical cases, 100, or even 200, grains are given at a dose. If the remedy be taken in a half pint of milk, or, better, in carbonated or aërated water, such as Apollinaris or Vichy, or even in the common siphon Seltzer, and after meals, there is rarely any difficulty even with the enormous doses indicated. Should a diarrheal tendency appear, it can be readily checked with bismuth preparations. Large doses of the iodids are much less likely to seriously disturb the patients than those of 7 to 15 grains. Mercury can be reliably exhibited by inunctions of 30 or 60 grains of the ointment daily. Various portions of the body, as the flanks, the back, and the extremities, should be used in rotation day after day. The danger of salivation can practically be eliminated if careful attention is given to the teeth and mouth. The use of mild antiseptic preparations, such as glycozone or boric acid, applied with a soft tooth-brush, and in gargles after taking food, is usually sufficient if the teeth are in good order; otherwise a dentist should be called in at once. The chlorate of potassium pastes and dentifrices are also useful.

The long-continued use of either iodids or mercury, singly or combined, is injudicious. After the first two or three weeks the mercury may be stopped and the iodid continued for another three weeks at such a dose as the progress or remission of the disease or the patient's tolerance indicates. It should then be discontinued and mercury may again be employed. It is clinically proved that the syphilitic process can gain such a tolerance for either mercury or iodid that their specific effect is lost. Hence the imperative need of intermissions or alternations in their employment. Personally, the writer does not feel safe in the treatment of these cases without the use of both remedies. By alternation they seem to be mutually helpful.

If the case progress favorably, the iodid for six weeks and the mercury for three weeks may be alternated in full doses for six months. Thereafter the patient should have a course of iodid and mercury every six months for five years, and finally once every year for the rest of life. Any reappearance of syphilitic activity should call for an immediate return to the intensive treatment.

In fighting syphilis we should never forget the patient. At times all specifics must be withdrawn and the general system built up. This is sometimes necessary where the limit of toleration appears to be reached and the symptoms of specific activity are still progressing. Supportive measures are indicated at all times. Ferruginous tonics, a liberal diet, massage, salt baths, and frictions are all valuable. The use of large quantities of drinking-water, hot or cold, and the employment of hot baths, or Turkish baths, if they can be borne, are of assistance and often enable the use of larger doses of specifics than could otherwise be exhibited. Here arise the benefits of thermal springs and similar resorts, at which, in addition, the patient is often freed from domestic and business worries.

In place of inunctions, sometimes the oral or hypodermic administration of mercury may be used, or plasters may be employed. Their reliability and efficiency are open to some misgivings, except in the case of injections. In cases of great urgency injections should always be employed at first. Bichlorid of mercury in doses of $\frac{1}{10}$ to $\frac{1}{2}$ of a grain may be injected into the muscles daily to secure prompt effects. The formula of Lambkin [1] has served well in a number of cases of cerebral and cord syphilis and in controlling the active syphilitic processes in tabes. Unlike injections of calomel and other insoluble salts, it rarely gives rise to local ulceration. It consists of one part of metallic mercury, one part lanolin, and one part of two per cent. carbolized olive oil, carefully triturated and sterilized on the water-bath. The dose is five to ten minims once a week into the muscles of the buttocks or loins, but there is decided danger of producing salivation. Mercurial baths are also available. Occasionally, the alterative vegetable compounds will be found valuable when mercury and iodids appear to have been exhausted. Slight looseness of the bowels does not contraindicate mercury, nor acne mean iodism. The slightest tenderness in the gums should lead to the immediate and complete cessation of mercury, to the use of which, with caution, the physician may usually return in a week.

The results of cerebrospinal syphilis in the way of plegias and nerve-atrophies require the same management as when arising from other causes.

THE PARASYPHILITIC DISEASES.

Aside from the ordinary luetic lesions of the brain and spinal cord, there is the long list of parasyphilitic diseases. These are not marked by round-cell invasion and gummy process, nor are they directly amenable to specific medication. They seem to be attributable to the toxic or chemical properties secondary to active infection, and may develop hereditarily, or, as is more commonly the case, follow acquired syphilis. A brief consideration is all that is required here, as they are individually dealt with elsewhere.

Acquired Parasyphilitic Diseases.—Chief among these are *tabes* and *paretic dementia*, which are practically always postsyphilitic accidents. In the cachexia of lues the general physical depravity furnishes a favorable soil for the development of *neuralgia*, *hysteria*, and *neurasthenia*, which do not differ from the same neuroses arising independently of syphilis, but they are benefited or cured by the removal of the specific cachexia. They are usually attended by considerable mental depression, the mental equivalent of the muscular asthenia.

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[1] "Brit. Med. Jour.," Feb. 19, 1898.

According to Fournier,[1] in addition to the epileptoid manifestations of cortical invasion, there is an epilepsy peculiar to syphilitics. Its characteristics he outlines as follows: (1) It is unattended by other evidence of cerebral disease; (2) it continues unchanged; (3) it is of long duration, even lasting the lifetime; (4) it is not amenable to antisyphilitic remedies; (5) the bromids have little control over it. Its onset is abrupt and usually in the form of a full epileptic seizure, without prodromata or inciting causes, and it often continues in the form of petit mal, grand mal, or a varying association of both. The attacks are commonly frequent during the first two or three years, after which they appear at long intervals only. Confirmation of this doctrine is lacking as yet, but cases corresponding to the outline are not extremely rare.

Hereditary Parasyphilitic Diseases.—In this category can certainly be placed the rare cases of *juvenile tabes* and *paretic dementia*. In addition all variations of *deficient vitality* and *defective growth* may be due to parental syphilis. Notably traceable to that source are *infantilism, mental defects, idiocy, hydrocephalus,* and various *cerebral and spinal agenetic states.*

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[1] "Les Affections Parasyphilitiques," Paris, 1894.

PART VII.

DISEASES OF THE NERVOUS SYSTEM WITHOUT KNOWN ANATOMICAL BASIS.

A LARGE number of diseases clearly related to the nervous system and manifested on its part by disturbance of functional control are not marked by known changes in the neural apparatus. They are denominated *neuroses* or *functional nervous diseases*. It is better to avoid the term functional, as it tends to distract attention from the probably existing anatomical basis of these diseases. There is little doubt that sooner or later they will be histologically classified. The present arrangement is for utility only. The following table serves to group the neuroses for descriptive purposes.

NEUROSES.

I. TROPHONEUROSES.—Marked by trophic faults and changes in physical conformation.	Acromegalia, Pulmonary osteo-arthropathy, Hyperostosis cranii, Myxedema, Exophthalmic goiter, Scleroderma, Raynaud's disease, Acroparesthesia, Intermittent limping, Angioneurotic edema, Localized hypertrophies.
II. INFECTION NEUROSES.—Mainly marked by motor disturbance.	Tetanus, Tetany, Hydrophobia, Chorea.
III. MOTOR NEUROSES.	Huntingdon's disease, Myoclonia, Dubini's disease, Parkinson's disease, Thomsen's disease.
IV. FATIGUE NEUROSES.	Writers' cramp, Occupation spasms, etc.
V. PSYCHONEUROSES.	Neurasthenia, Hysteria, Epilepsy, Migraine, Tics.
VI. NEUROSES FOLLOWING TRAUMATISM.	

CHAPTER I.

TROPHONEUROSES.

IN the group of neuroses marked by disturbances of nutrition some abnormality of the vasomotor control is commonly present. This may furnish nearly the entire symptomatology, as in angioneurotic edema, Raynaud's disease, and exophthalmic goiter. In other instances the vascular element is apparently small, and the processes of nutrition are principally disordered. The relation of nutrition to vascularity, however, is always intimate. We can not as yet positively say through what individual strand of fibers trophic control is maintained, but it must be in close touch functionally with the vasomotor nervous apparatus.

ACROMEGALIA.

In 1886 P. Marie[1] called attention to two cases presenting acquired symmetrical enlargement of the hands, feet, and face, and proposed the name acromegalia, which has now become current. His description of these cases was so full that little has since been added to the clinical side of it. Similar cases were at once recognized all over the world. In 1892 Collins[2] was able to collect about ninety cases from literature. Since then the material has rapidly increased, and many autopsies have been put on record.

Etiology.—The causation of the disease is extremely obscure. Alleged or supposed inciting causes are almost as numerous as recorded cases, and embrace well-nigh every ordinary and exceptional experience of human life; consequently, no importance at present attaches to any of them. Both sexes are about equally affected. The disease makes its appearance customarily between the ages of eighteen and thirty. Exceptional cases have developed in advanced life and others in childhood. Several have appeared so early that they have been considered congenital. It has been found in association with tabes, syringomyelia, rachitis, gigantism, exophthalmic goiter, goiter, and various psychoses.

Klebs, impressed by his first case in which the thymus gland persisted, and Erb by the area of sternal dullness in other cases, were disposed to consider the changes in the bones and soft parts as due to a thymic angiomatosis. Later cases failed to present the enlarged thymus, and the sternal dullness is probably sometimes due to thickening of the bone itself. Some, with Freund, have attributed the somatic changes of acromegalia to inversion in the evolution of genital life. Recklinghausen considered it an angioneurosis dependent upon central changes. These changes are not found, and angiomatosis is not evident in the tissues.

The usual adenomatous enlargement of the pituitary body and the frequent defective or goitrous condition of the thyroid have led to the belief that acromegalia is a trophoneurosis dependent upon modifications of the functions of these glands, especially of the pituitary. Rogowitsch, for example, considers that these glands destroy certain substances which have a toxic influence on the central nervous system. Others think that they secrete certain substances needful to the proper action of the trophic apparatus. All that can now be said is that the prehypophysis cerebri is nearly if not always hypertrophied, but whether as a cause or result is not determined. It is true, however, that tumors and other destructive conditions of the pituitary have been found without acromegalia.[1] Caselli contends that the pituitary gland is essential to life in dogs and cats, while Friedman and Maas[2] deny it. Israel[3] reports a well-marked case of acromegaly under observation for some years in which the pituitary gland was normal.

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[1] "Revue de Méd.," 1886.

[2] "Jour. Ment. and Nerv. Dis." N. Y., 1892.

Morbid Anatomy.—The *bones* of the face, cranium, extremities, and to a lesser degree those of the trunk, show hypertrophy. The frontal and occipital bones are commonly thickened and their markings exaggerated. The frontal and maxillary sinuses are enlarged and the pituitary fossa is greatly increased in size. The inferior maxilla, the malars, the zygomatic arches, and the superciliary ridges of the frontal are particularly enlarged. There is commonly elongation of the spinous processes in the cervicodorsal region, and the bodies of the vertebræ are sometimes increased in the anteroposterior diameter. The ribs, clavicles, and sternum are commonly, and the shoulder-blades occasionally, enlarged. In the limbs the hypertrophy is most marked toward the distal extremities. The long bones of the leg and forearm are most changed at their lower ends. The metacarpals, metatarsals, and phalanges are particularly involved. Histologically, the hypertrophy is a true one, bone being deposited under the thickened periosteum and the central canal enlarged by the action of osteoblasts.

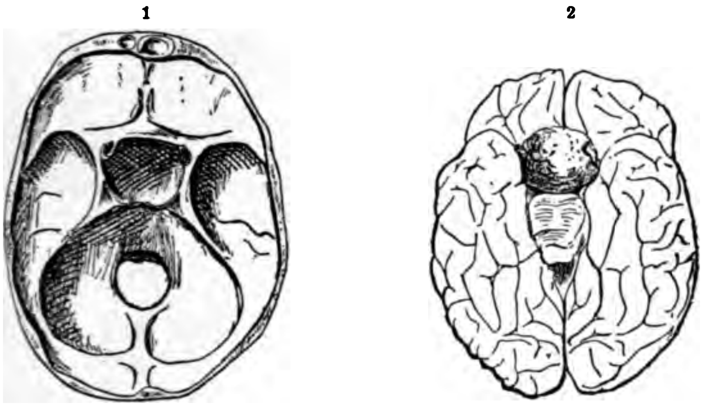

Fig. 210.—1, Base of cranium showing excavation of the sella and, 2, pituitary tumor in a case of acromegalia (Marie).

The *pituitary gland* is enlarged and hypertrophic, filling up the distended and enlarged sella turcica and commonly compressing the optic chiasm and adjoining structures. It practically constitutes an endocranial tumor. Strümpell[1] is inclined to think it an essential finding. In many cases it has been found to have undergone changes, probably secondary in point of time and nature. Tamburini[2] believes the first stage is one of enlargement and increased function, followed by adenomatous, sarcomatous, or cystic degeneration. A number of cases showing such degenerative changes are cited by Strümpell[3] and reported by others.

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[1] Jokoloff, "Virchow's Archiv," Bd. xliii.

[2] "Berlin. med. Woch.," 1900.

[3] "Virchow's Archiv," vol. clxiv, 344.

In the extremities and other portions affected by the hypertrophic enlargement the *skin* in its epithelial, dermal, glandular, and muscular parts is hypertrophically exaggerated. The connective tissue particularly is increased. The subcutaneous nerve-filaments are sometimes degenerated as a result of the fibrous hyperplasia, but this does not extend into the nerve-trunks. The skin is frequently overactive and the perspiration may be excessive and disagreeably fetid. In color the skin is usually muddy, especially toward the extremities, and frequently marked with warts and pendulous, soft fibromata. The cutaneous *sensibility* is practically normal, but these patients are usually very sensitive to cold. The *mucous membrane* of the nasopharynx, larynx, and tongue are similarly affected. The *kidneys* often show a moderate chronic parenchymatous nephritis, with interstitial fibrosis. The *spleen* and *lymphatic glands* may be sclerosed; the *thyroid* is usually atrophic; the *thymus* may persist and even be enlarged. It is to be noticed that, while parenchymatous glandular structures generally are atrophic, the pituitary is hypertrophic.

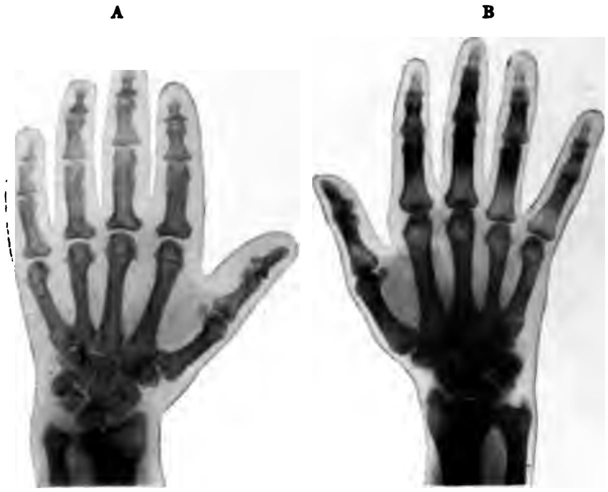

Fig. 211.—Skiagraphs of hands: A, Case of acromegalia; B, normal hand.

Symptoms.—Among the most common symptoms of acromegalia we may name hypertrophy of the face, hands, and feet, visual defects, dorsal kyphotic deformities, persistent headache, sexual failure in men, and amenorrhea in women.

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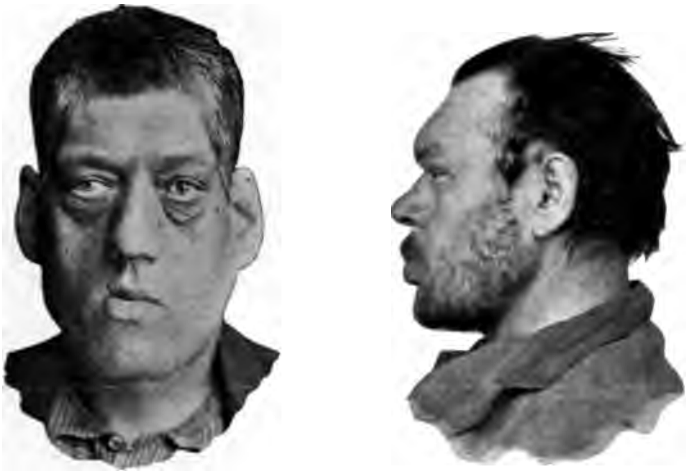
[1] "Deutsche Zeit. f. Nervenheilk.," 1897.

[2] "Centralblatt f. Nervenh.," Dec., 1894.

[3] *Loc. cit.*

The *face* is notably deformed. The greatly enlarged, overhanging brows are marked by shaggy, coarse eyebrows. Thickened eyelids; sometimes *exophthalmos;* a heavy, large, flabby *nose;* great *cheek-bones;* an enormous *mouth,* correspondingly thickened *lips,* and a much enlarged and prognathic *lower jaw* are some of the facial characteristics. The heavy features, puffy face, and accentuated nasolabial folds give an *expression of grief and suffering.* When the mouth is opened, it is seen that the hypertrophic enlargement of the *jaws,* especially of the mandible, causes the *teeth* to stand separately and at considerable intervals. The *mucous membrane* is also thickened and the *tongue* is notably enlarged, in some instances reaching an enormous size. The soft palate, pharynx, and larynx equally share in the hyperplasia, and the tonsils and follicular structures are also increased in size. The ears and occiput are sometimes enlarged, sometimes not. The *scalp* is thickened, the *hair* coarse, thick, and harsh. The *complexion* is usually sallow and the parts are elastically *doughy* to the touch, showing no pits on pressure.



Figs. 212 and 213.—Face in acromegalia, two cases. Note enlarged superciliary ridge, thickened lips, massive jaw, and general grossness.

The *hands* are greatly enlarged in all their dimensions, but particularly in width. The *proportion* of fingers to hand and hand to wrist is retained; they are, therefore, enlarged symmetrically. The *fingers* are thick and sausage-shaped, the hand thick and beefy, the thenar and hypothenar eminences greatly but proportionately enlarged. The whole hand is described as "spade-like or battledore-shaped." This appearance is sometimes intensified by a comparative thinness of the forearms. The *joints* are never limited in their range of motion, and the parts are surprisingly supple and flexible. The *palmar furrows* are usually much deepened, the *skin* thick and resilient, the hairs coarse, the *nails* broadened, thick, and strongly striated longitudinally.

The *feet* show similar changes, and usually there is a heavy welt of fibro-elastic increase around the heel and along the outer border of the foot.

The *thorax* usually presents an *anteroposterior curvature* in the cervicodorsal region, which may be compensated by lumbar lordosis and attended by scoliotic twists. The *clavicles* are almost invariably increased in thickness and sometimes in length, and the *sternum* may also share in the hypertrophy. The *ribs* and *cartilages* are often enlarged. These bony changes give the chest an unusual breadth and anteroposterior depth. The *respiration* is ordinarily abdominal in character, and the abdomen is often protuberant or even pendulous. The *pelvic girdle* may be enlarged.

The external *genitalia* in women are ordinarily hypertrophied, the uterus atrophic. In men there may be hypertrophy or atrophy of the external genitals. *Amenorrhea* and *sterility* are the rule with women, sexual inappetence and *impotence* with men.

Cephalalgia of an intense, persistent, deep-seated character is present in the great majority of cases.

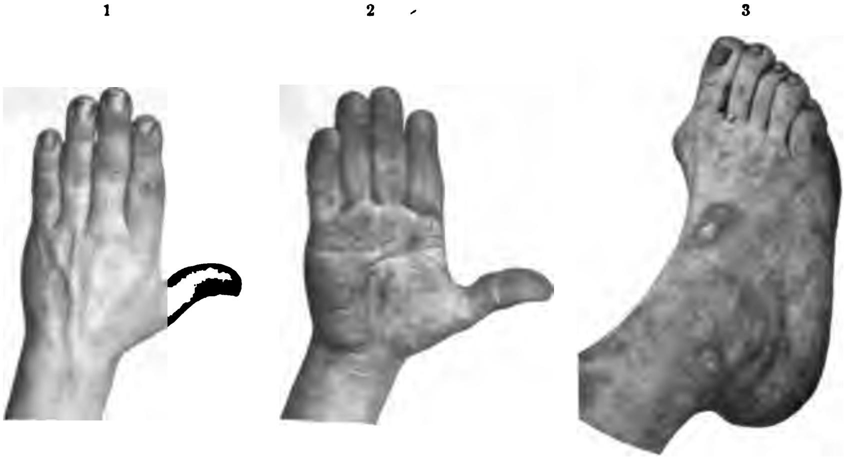


Fig. 214.—1 and 2, Hand in acromegalia; 3, foot in acromegalia.

Speech is frequently thickened from the clumsiness and voluminous size of the tongue, and the voice is ordinarily very deep, strong, and rough, owing to the enlargement of the *larynx*, which to palpation may appear of increased dimensions and unusual prominence.

The *organs of special sense* may all be affected. *Smell*, *taste*, or *hearing* may be reduced or abolished, but this is uncommon. *Vision*, however, is usually more or less affected eventually. This may arise from *optic neuritis* or from *atrophy*, and in either case points to encephalic tumor. Amblyopia, blindness, intra-ocular pains, exophthalmos, contraction of the visual field, temporal hemianopsia, and miosis have been noted. The bilateral loss of the temporal fields or their manifest reduction indicates injury to the chiasm by the pituitary tumor.

The *muscular system* may be normal or some atrophy may be found, but general muscular weakness is the rule. The *reflexes* are normal or diminished. The *electrical responses* are frequently quantitatively reduced. Cardiac hypertrophy, arteriosclerosis, varicose veins, and slight lymphatic adenopathy may be encountered. The *thyroid* may be absent, normal, or hypertrophic. Polyuria, glycosuria, polydipsia, excessive appetite for food, and dyspepsia are common.

General *physical feebleness* is usual, and *mental sluggishness*, irritability, and inaptitude are common.

Course and Forms.—The disease is of insidious *onset* and slow progression, presenting a duration of twenty to thirty years and terminating by a cachectic state marked by great muscular weakness, during which death often occurs suddenly through cardiac failure. Intercurrent affections find acromegalics very vulnerable.

We may distinguish *two varieties* of acromegalia, depending mainly upon the age at which the disease commences and the condition of epiphyseal ossification with the diaphyses: (1) Before complete ossification takes place the enlargement is not only in lateral dimensions, but also in length, and the limbs become disproportionate to the body, producing *gigantism.* Autopsical findings by Hutchinson, Dana, Bramwell, Taruffi, and skiagraphical investigations by Marinesco conclusively show this relation. (2) After the diaphyses and the epiphyses are united by bone, only circumferential increase is produced, and the stature, from scoliosis, may actually be reduced. The commoner *massive type* originally described is then developed.

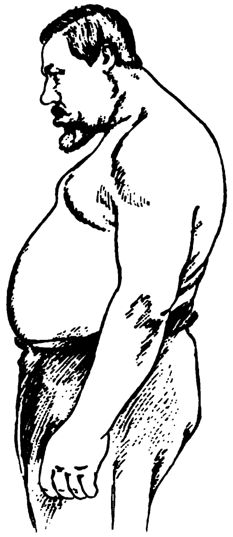


Fig. 215. — Cervicodorsal curvature of spine; chest and abdominal distortion in acromegalia (Marie).

Diagnosis.—The diagnosis can rarely present difficulty if the disease has attained any considerable degree of development. *Myxedema* shows no osseous deformities, and the thickening of the soft parts is waxy and boggy. *Pulmonary osteo-arthropathy* presents joint-changes and a chronic pulmonary process. The clubbed enlargement of the fingers is largely confined to the terminal phalanges, over which the hypertrophic nails hang like parrot-beaks. In *hyperostosis cranii* the hands, feet, and mandible are not affected. Paget's disease, or *osteitis deformans*, spares the face, but affects the skull, and causes bowing of the long bones, especially the femora and tibiæ. The x-ray may show excavation of the sella.

Prognosis.—The progress, as above indicated, is toward physical helplessness and mental depression, which in more than one instance have led to suicide. According to Thompson,[1] the disease may run an acute, intermittent, or chronic course, lasting from two to thirty years.

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[1] "Brit. Med. Jour.," April 9, 1899.

Sudden death from cardiac failure is rather frequent. As yet the disease is not therapeutically manageable.

Treatment is confined to relieving the cephalalgia and correcting the symptomatic disturbances as far as possible. For the first, phenacetin and similar synthetical anodynes seem to be most efficacious. Treatment by thyroid, thymus, and pituitary preparations, singly or combined, seems to be of little avail, except in some instances to reduce weight, and then often at the expense of the general health and strength. Animal extracts may, however, yet control the malady, but a recession of osteal hypertrophy is too much to reasonably anticipate when once it has developed.

HYPERTROPHIC OSTEO-ARTHROPATHY.

In 1890 Marie and, independently, Bamberger described cases of chronic intrathoracic disease attended by skeletal deformities. For such, Marie proposed the term *osteo-arthropathie hypertrophiante pneumique.* Massalongo, Thayer, and others insist that the pulmonary factor is by no means essential, and Thayer [1] found it absent in 12 out of 55 typical cases. Other chronic conditions to which it is secondary are syphilis, heart disease, diarrhea, and spinal caries. It has been associated with phthisis, empyema, bronchiectasis, pulmonary syphilis and neoplasms, pleurisy, bronchitis, and gastric dilatation. The disease presents a multiple osteitis, affecting mainly the terminal phalanges of the hands and feet, the extremities of the long bones of the limbs, and the neighboring joints. The affected bones are enlarged, rarefied, and show the evidence of inflammation. The cartilages are eroded, the synovial membranes thickened. The bones, however, may escape,[2] the enlargement of the finger-ends being due to an excess of subcutaneous fat. The fingers, owing to the globular enlargement of the distal phalanges, have been likened to drumsticks. The nails are greatly enlarged and overhang the thickened finger-ends like a parrot's beak. Usually they are thin and smooth. The joints creak and grate on motion, and the ankles and wrists are ordinarily broadened and thickened. Elbows and knees may be similarly affected, and the toes show deformities and disproportions similar to those of the fingers. The invariable presence of chronic toxemic states indicates a toxic basis for the trophoneurosis; for instance, Preble [3] noted it in a case of gastrectasis.

The disease usually is of insidious onset and is frequently attended by rheumatoid pains. It may occur at any age.

The diagnosis is commonly easily made, though at first this dystrophic disease was confused with acromegalia. It does not affect the head, face, or soft parts, and does involve the joints. In acromegalia the fingers and toes are uniformly enlarged, and the wrists and ankles are not disproportionately increased in size. The facies of acromegalia is never seen in pulmonary osteo-arthropathy.

The disease runs a protracted course, but may come to a standstill, or it may recede slightly if the thoracic condition is greatly improved. Desmons and Beraud [4] report some improvement following subcutaneous injections of extracts made from sheep's lungs.

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[1] "Phila. Med. Jour.," Nov. 5, 1898.

[2] E. F. Buzzard, "Br. Med. Jour.," June 1, 1902.

[3] "Medicine," Jan., 1898.

[4] "Archives gen. de Méd.," 1894.

HYPEROSTOSIS CRANII.

Virchow used the term *leontiasis ossea*, Starr, *megalocephalie*, and Putnam, *hyperostosis cranii* to designate certain rare cases in which the bones of the cranium, and less markedly those of the face, undergo enormous enlargement. The process is apparently inflammatory in character, but its symmetrical distribution, association with developmental periods of life, and progressive nature point to a lack of trophic control. Putnam,[1] accepting seven from Baumgarten's list, has been able to tabulate fifteen cases, including that of Edes. Morton Prince[2] believes that this condition is but a part of osteitis deformans, with which it is sometimes associated, and that both are of neuropathic origin. In the majority the disease appears under thirty years of age, and often in childhood or at puberty. The cranial enlargement is sometimes preceded by inflammations about the head, such as erysipelas, and by traumatism. Early symptoms have been headache, drowsiness, epileptic attacks, deafness, and blindness. Mental irritability or enfeeblement is common. Exophthalmos, loss of hearing, facial palsy, optic neuritis, and blindness are due to local or intracranial pressure. The anterior portion of the head is usually most enlarged, and the upper

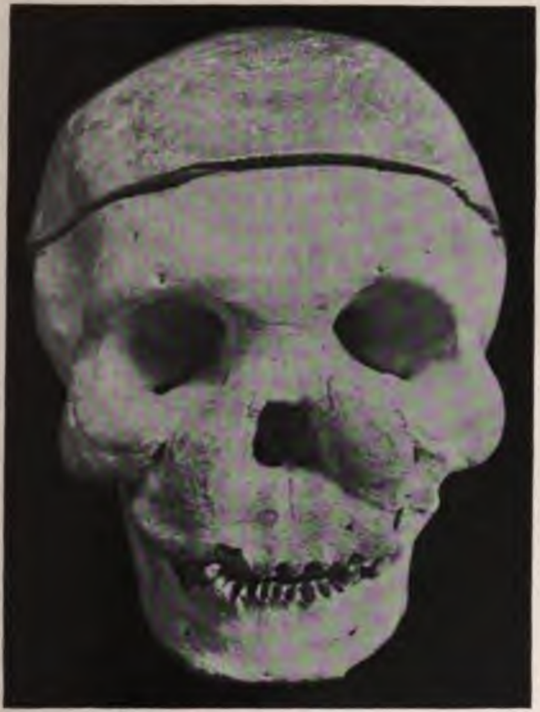

Fig. 216.—Hyperostosis cranii (Putnam).

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[1] "Amer. Jour. Med. Sciences," July, 1896.
[2] "Amer. Jour. Med. Sciences," Nov., 1902.

facial bones are more affected than the inferior maxilla. Prognathism is not commonly observed. There is usually a large increase in the bitemporal diameter. In some instances the bones are rather evenly enlarged; in others there are numerous exostotic thickenings, both on the outer and inner surface of the cranium, or on either aspect alone. The cervical vertebræ are sometimes also enlarged. The disease is progressive and not amenable to treatment. Putnam, Starr, and others have tried thyroids in vain. Encephalic pressure may in suitable cases be relieved by trephining.

MYXEDEMA.

Under the generic term of myxedema it is proposed to bring together those clinical variations of nutritive disorder dependent upon partial or complete cessation of thyroid activity. In 1873 Gull described a *cretinoid state occurring in adult women*. Four years later Ord reported additional cases and proposed the word *myxedema* (mucous swelling). Charcot, about this time, struck by the cutaneous thickening and the cachectic state, used the term *pachydermatous cachexia*. In 1880 Bourneville and d'Olier described a case of myxedematous idiocy and subsequently many more. In 1882 Reverdin, and shortly afterward Kocher, reported myxedema following complete extirpation of the thyroid, for which they severally proposed the terms of *operative myxedema* and *cachexia strumipriva*. The analogies between acquired myxedema, myxedematous idiocy, and *cretinism* have been noted by many, and Brissaud, in his lectures of 1893–'94, brings certain cases of *infantilism*, or physical retardation, into the same category. We will commence by a description of acquired myxedema in adults. Due allowance being made for the age and growth of the individual, and the suddenness and completeness with which the thyroid is affected, will practically enable this description to be applied to all the above-mentioned varieties.

Acquired myxedema of adults is of insidious onset, as a rule, and more common in women than in men. It usually appears between the ages of thirty and fifty. Occasionally it has abruptly followed an attack of acute rheumatism, some infectious fever, or a severe hemorrhage. It is marked by: (1) Tumefaction of the dermal and mucous structures; (2) by intellectual and physical enfeeblement, and (3) by atrophy of the thyroid body.

The *dermal changes* are the most striking. The skin is infiltrated with a mucoid substance and the fatty panniculus is frequently greatly thickened. The swelling offers an elastic resistance to the touch and does not pit on pressure. The color is usually pale, yellowish, and waxy or cheesy. The face is enlarged, rounded, and apathetic. The heavy, thickened lids droop over the eyes, the nose and lips are thickened, the brow is furrowed, the ears enlarged, and the cheeks rounded and showing jelly-like trembling on slight jarring. The whole appearance is one of hebetude and stupidity. The skin of the trunk and extremities is likewise infiltrated. In the axillæ and subclavicular depressions lipomatous-like masses are often encountered. The hands and feet are enlarged with cushion-like swellings on their dorsal surfaces; the digits are thick and clumsy. The scrotum is often much thickened. The *epidermal structures* are greatly affected. The skin is dry, harsh, and branny. The hair becomes dry and scanty on all portions of the head and body. The nails are dry, fragile, striated, atrophic. Perspiration and sebaceous secretions are defective. The *mucous membranes*, wherever visible, are tumefied, pale, dry, and elastic. Mucous polypi in the nasopharynx are not uncommon. The *tongue* is thickened and, with the infiltrated condition of the buccal, pharyngeal, and laryngeal mucous membranes, explains the muffled voice and difficulty of swallowing.

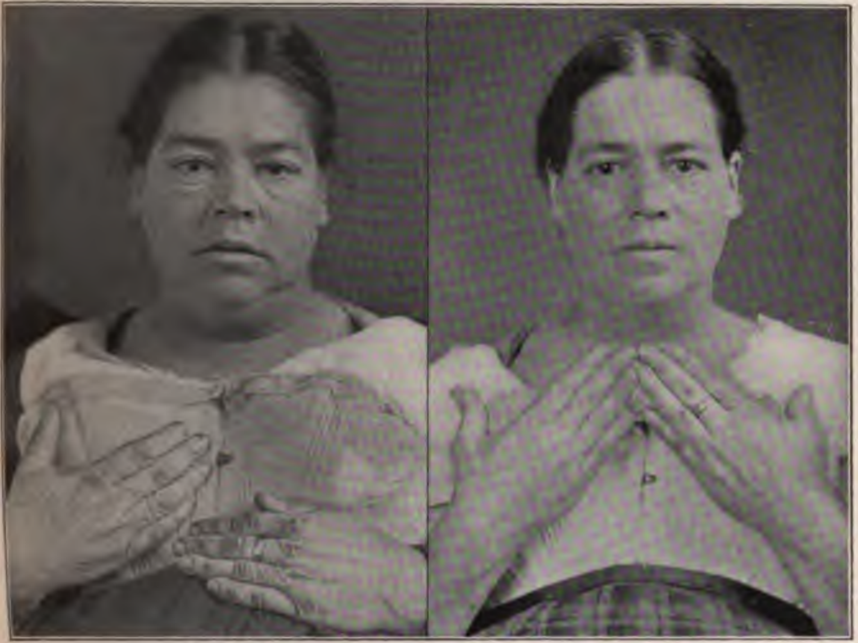

Fig. 217.—A case of myxedema before and after two months' treatment by thyroids (Dr. John Woodman).

The *intellectual state* in myxedema is uniformly marked by apathetic enfeeblement, and cerebral torpor shows itself in sluggish mentation, defective memory, slow speech, and lethargic movements. Myxedemic patients are usually indolent both mentally and physically, and are irritable and somnolent. Some cases show nocturnal insomnia and are troubled by disturbing dreams. The sluggishness of movement and the clumsiness of the hands and feet are due to the cerebral torpor and the local thickenings. These patients have no muscular energy and are promptly fatigued on the slightest continuous effort, but there is no palsy.

The *thyroid* in the great majority of cases can not be detected by palpation. There may be a history of its former presence or actual enlargement, and even a goitrous condition may persist. In some instances exophthalmic goiter has preceded myxedema and they have also been found associated, but Graves' disease does not follow myxedema.

Less prominent and constant conditions in myxedema are: cardiac weakness, irregular pulse, hemorrhages (especially metrorrhagia), a subnormal temperature, loss of teeth, habitual constipation, and occasionally albuminuria in advanced cases. The patients complain of headaches, vertigo, throbbing in the ears, and particularly and almost constantly of a sensation of cold. Sensations objectively, motor conditions, and the reflexes are normal.

Usually insidious in *onset*, the disease runs a slow, tardy, progressive course. Remissions of longer or shorter duration may occur, as in summer, or by removal to a warm climate, and pregnancy sometimes has a similar retarding effect. The general tendency is toward cachectic helplessness and death by marasmus. In the very last stages the tumefaction may disappear. Pulmonary complications, especially tuberculosis, are common, and cardiac asthenia may strike the final note. Fortunately, treatment is now equal to the requirements of these otherwise hopeless cases.

Operative myxedema is usually the result of the total extirpation of the thyroid, which it follows in from three to six months. Removal of a part of the thyroid, the remainder being completely disabled,—by cystic disease, for instance,—results in myxedema. This is initiated by lassitude, physical enfeeblement, sensations of cold, heaviness in the limbs, sluggish and clumsy movements. Shortly the integument tumefies and becomes discolored, the hair falls, and cutaneous functions lag. The cerebral torpor follows and myxedema is fully established. Tetany may also develop, due, according to Murray,[1] to the loss of the parathyroid bodies, which in man are included in the lobes of the thyroid. The course is usually progressive, but is more subject to remissions than in the spontaneous variety. Other cases improve, owing to the compensatory action of unremoved portions of the gland, or to the development of accessory thyroids, or through the vicarious activity of other glandular structures. The gravity of operative myxedema is great in proportion as the patient is young. Occurring in childhood, or at any period before adult life, it retards or completely checks growth, and produces a persistent infantilism or a myxedematous idiocy. It is, however, completely amenable to the thyroid treatment.

Congenital myxedema, myxedematous idiocy, or sporadic cretinism is usually first noticed at about one year of age, or upon weaning, and thereafter presents all the characteristics of adult myxedema, excepting that the mental faculties never develop and physical growth is retarded to the last degree. It is frequently congenital, and Horsley has found it in a dead-born fetus. It is encountered in the offspring of degenerate, alcoholic, or phthisical parents. At twenty years of age these cretinoid idiots may be of little more than two feet of stature. The relatively normal size of the head contrasts with the dwarfish body. The skull is full behind, contracted and narrow in front, often with persistent fontanel. The flabby, thickened features; snubbed nose, thick lips, drooping eyelids, mouth agap, lolling, hypertrophic tongue, and drooling saliva make up a peculiarly repulsive appearance. Add, now, the short, often lipomatous, neck; a protuberant abdomen, often showing inguinal and umbilical ruptures; a deviating spine, rudimentary genitals, and dwarfish, crooked limbs, and it is impossible to imagine a less human-looking object with human attributes. Sparse hair, eczema, and an infiltrated, inactive skin are commonly present. The thyroid is absent. Idiots mentally, they can ordinarily exercise a little attention and even show some affection. In some instances they assist themselves in eating and dressing. They never learn to speak, and never show signs of pubescence. Often even the first dentition is extremely defective. On the other hand, they lack the destructiveness, noisiness, tics, convulsions, onanism, balancing and motor disturbances so common in idiocy from cerebral lesion. These unfortunate creatures may attain thirty or forty years of age, and usually die of pulmonary concomitants.

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[1] "Br. Med. Jour.," Mar. 18, 1899.

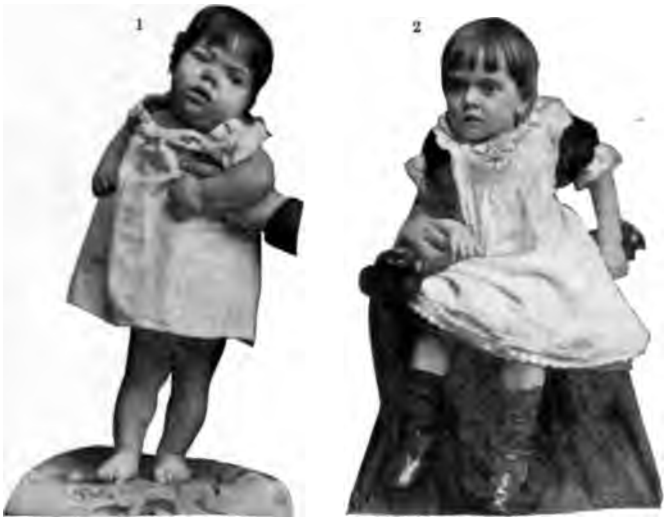

Fig. 218.—Sporadic cretin before and after twelve months' thyroid treatment: 1, Five years old; 2, one year later (Parker).

The disease is also amenable in some degree to the thyroid treatment.

Cretinism is a term of ancient lineage and honored usage, but of somewhat uncertain definition. It has been applied to the goitrous and feeble-minded natives of localities where goiter is endemic. Certain valleys in France, Spain, Italy, and Switzerland, and some parts of Great Britain, Sweden, and of other countries widely scattered over the globe present endemic conditions that predispose to goitrous enlargements. Such an endemic has been noted in Minnesota and Ontario. A few definite facts are the results of observations extending over generations in some of these communities. The offspring of two goitrous parents, according to Kocher, is invariably a cretin, who may or may not be goitrous, but is myxedematous. A non-goitrous cretin invariably has goitrous antecedents and is indistinguishable from the myxedematous idiot or sporadic cretin, the condition also being congenital. In the goitrous cretins the thyroid disease may appear at any period of life, and acts then, exactly as does spontaneous myxedema or operative myxedema, to stunt growth and stop mental development. The goitrous cretin is usually also myxedematous and may present any degree of mental impairment, from mere simple-mindedness to abject brutishness. The distribution of endemic cretinism is identical with that of endemic goitrous disease, and Kocher believes this to be due to organic infections through the water-supplies.

The only distinction between endemic cretins and other myxedemic patients is the goitrous enlargement. This may be only a difference of degree, because the cystic degeneration and interstitial hypertrophy at the bottom of the goitrous enlargement of the thyroid is destructive in character and effect. It is easily conceivable that myxedema and mental disturbances will be developed proportionately to the lack of functionally active thyroid. When the thyroid is entirely wanting, as in the non-goitrous congenital cretin, or completely destroyed in some goitrous cretins, the myxedema is correspondingly intense and the mental degeneration proportionately developed. It would seem, therefore, that the causes at the bottom of endemic cretinism are those that produce endemic goitrous disease, to which the cretinoid state is secondary. These are not well understood, but usually the soil, drinking-water, and atmospheric conditions are held accountable.

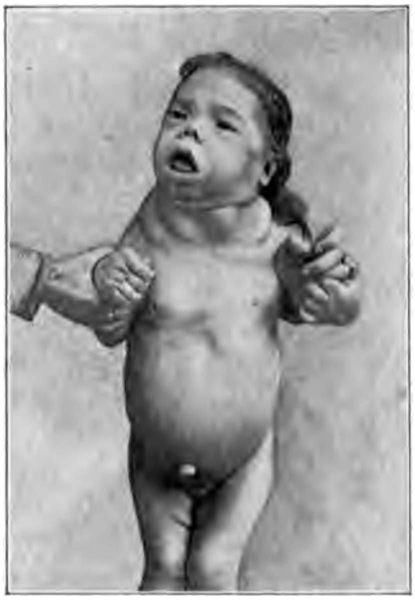

Fig. 219.—Myxedematous cretin eighteen years old (Parker).

Infantilism and Myxedematous Retardation.—Occupying a middle ground between myxedematous idiocy and acquired myxedema there are numerous cases showing slight cutaneous tumefaction, retarded mental development, and diminished growth. These patients retain their childishness both in mental attributes and physical conformation. Perhaps here belong some of the idiots classed as Mongolian by the English, and some of the *enfants aricarré* of the French. In at least one well-marked instance Brissaud found the thyroid body scarcely perceptible. At the age of ten numerous and large cervical glands had been removed with probable resulting damage to the thyroid. Thereafter the physical, genital, and mental growth of the lad had remained stationary. Schmidt[1] has treated three cases of this sort in which growth was retarded, by the administration of thyroids, with immediate improvement.

Etiology.—If we look upon myxedema as the manifestation of defective thyroidation, its causes are those of disease or absence of the thyroid body. In some cases it is a *teratological defect;* in others it is the result of a *thyroiditis* which may be dependent on infectious processes, as the infectious fevers, rheumatism, etc.; in others it is cystic *degeneration;* in others the result of *trauma* or surgical *extirpation.* Taking all causes of myxedema together, there is a preponderance of *females* which reaches large proportions in the spontaneous adult varieties. It is probable that the close relation of the thyroid and uterine functions is at the bottom of this fact. It is only necessary to mention the increased size of the thyroid in pregnancy, its frequent enlargement in pubescent girls, its usual congestion in some women during menstruation, and its final retraction at the menopause. *Erysipelas* of the neck and head and *syphilis* of the thyroid gland have induced myxedema.

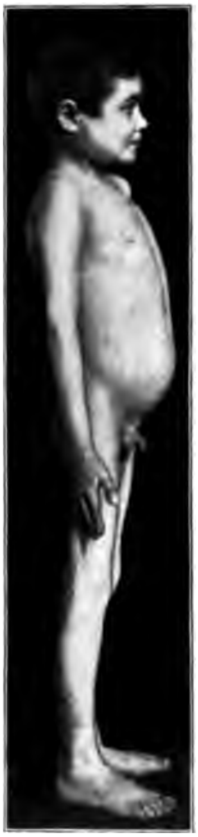

Fig. 220.—Myxedematous infantilism, youth nineteen years old (Brissaud).

Regarding the manner in which defective thyroidation affects the trophic apparatus and induces the mucoid deposits, two general points of view depending upon opposite physiological hypotheses are maintained. As yet all is theory. Many, with Schiff, believe that the normal thyroid elaborates some substance indispensable to the proper action of the nervous system. This substance, however, has never been isolated nor its characteristics determined. Others, with Horsley, conceive that the thyroid transforms the mucinoid elements of the blood into utilizable metabolic constituents, or, as a modification of this idea, that the thyroid eliminates certain harmful elements from the blood. The blood in myxedema is poor in oxygen and the urine is of an increased toxicity. Both of these conditions are favorably modified, as is the myxedematous state, by the administration of thyroids. It now is determined that the thyroid gland is essential to life and to the proper neurotrophic balance. That it is essentially secretory is proven by its embryonal duct in man opening at the root of the tongue and its active connection with alimentary processes in the lower orders.

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[1] "Therapeutische Wochen.," Nov., 1896.

Morbid Anatomy.—The primal lesion of myxedema is located in the thyroid. In myxedematous idiocy the gland is either wanting, rudimentary, or atrophic. In acquired myxedema it is atrophic, yellowish white, and fibrous. At first there appears to be an embryonal vesicular infiltration, with epithelial proliferation. Later, fibrous changes predominate, and finally complete sclerosis is presented. This practically constitutes a parenchymatous and interstitial thyroiditis. Murray, however, believes that atrophy of glandular tissue is the first step, due probably to the action of some toxic agent, and that the increase of fibrous tissue is a replacement-fibrosis. The cystic degeneration of cretinoid goiter reaches the same results by its destructive action upon the glandular parenchyma.

The *subcutaneous cellular tissues* are infiltrated with a gelatinous, mucinoid substance and the *fatty panniculus* is much increased. Throughout the organism there is a tendency to *fibrous proliferation* which especially affects glandular organs, as those of the skin and the kidneys. *Mucin* has also been found in the blood and in all the tissues of the body. The *pituitary* gland and the *thymus* are frequently enlarged, apparently for the purpose of compensation. The nervous system presents no changes that are uniform or significant. Central hyperemia and tumefaction of the nerve-cells have been noted by Rogowitch.

Treatment.—The treatment of myxedema furnishes one of the most brilliant chapters in medicine. It was observed that in animals and man myxedema did not follow partial ablation of the thyroid, and Schiff found that a portion of the gland subcutaneously or intraperitoneally implanted prevented the myxedematous state in animals subsequently thyroidectomized. Horsley suggested the same procedure in man, and it was carried into effect by Kocher, Lannelongue, Bircher, and others with favorable results. When the thyroid graft was successful, myxedema was temporarily checked. Murray first used subcutaneous injections of thyroid extracts or emulsions with better and more persistent results, and a series of cures were reported by this method in the hands of numerous observers. Howitz, of Copenhagen, followed shortly and independently by Mackenzie and Fox in England, and then by a host of physicians the world over, fed myxedemic patients with thyroids raw, partially cooked, or desiccated. The results were almost uniformly good. The procedure is perfectly justified not only by clinical results, but by the fact that in lower animals and in the human embryo the thyroid gland furnishes a secretion to the alimentary canal by a duct opening at the foramen cecum on the base of the tongue. The sheep's thyroid is habitually employed and is now an article of commerce in the form of various powders and tablets. The equivalent of one-half a sheep's thyroid may be administered daily, and if well tolerated, increased to a full thyroid or more. If prompt action is secured, it may be reduced, and if difficulties arise, it must be discontinued and resumed with caution. The exhibition of thyroids is followed in a day or two by a return of the temperature to the normal standard, which it may even exceed by a degree or two; the pulse becomes more rapid, the urine increases in volume, the pigmentary infiltration diminishes, the fatty deposits melt away, and the patient becomes more animated and cheerful. Four to eight weeks may show a marvelous change. The skin becomes softened and moist, the hair and nails are better nourished, and the normal state of the patient is shortly attained. To maintain the cure, thyroid feeding must be maintained. If the treatment is discontinued, the patient promptly relapses. The equivalent of one thyroid a week is usually sufficient for the purpose, and cases are now on record in which several years have been passed in apparent health and vigor under this régime.

The treatment is attended by considerable *danger*, and in some instances it has been followed by fatal results. Headaches, pains in the bowels, diarrhea, neuralgia, insomnia, malaise, and nervous excitement are some of the unpleasant symptoms which should promptly lead to a reduction of the dose or to the temporary interruption of the treatment. When the terminal cachexia of myxedema has appeared, this treatment is hazardous, but nothing else can possibly rescue the patient, and in some such cases it has done so. In children, especially in myxedematous idiots with rachitic deformities, thyroids tend to produce a softening of the bones that may greatly exaggerate the osseous distortions. Parker[1] and others have met with this complication, and have recommended that such cases be treated in bed, and the weight kept off the legs.

In acquired or operative myxedema thyroid feeding may be considered established as a curative treatment. In cases of myxedematous retardation its results appear equally brilliant. In sporadic cretinism it is capable of producing the most marvelous improvement, which is complete in proportion as it is adopted early in life. In endemic cretinism its effects are beneficial, but as yet its proper value is not established.

The action of thyroidin, a chemical compound isolated by Baumann,[2] is practically the same as that of the various preparations of the gland, but is less likely to be attended by the accidents of ptomain poisoning. Against the tetany that sometimes follows removal of the thyroid, it is less efficient than the thyroids themselves.

EXOPHTHALMIC GOITER.

Exophthalmic goiter, otherwise known as *Graves' disease*, *Basedow's disease*, *cardiothyroid exophthalmos*, and *struma exophthalmica*, presents, in most respects, a complete contrast to myxedema, but also shows some analogous features. We have considered myxedema as due to defective thyroidation, and we may, at least tentatively, look upon Graves' disease as the manifestation of excessive or perverted thyroid activity, or of both. The disease is manifest by the three so-called cardinal symptoms of rapid heart, enlarged thyroid, and prominent eyeballs. To this must be added a fine tremor, mental irritability, and muscular weakness, which are equally common. Further, nearly every function of organic life may be disturbed. Flajani, in Italy, described the condition in 1802, and Parry, in England, in 1825. Graves taught it as a disease entity in 1835, and published it as such in 1843; meanwhile Basedow, in Germany, had independently written of it in 1840.

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[1] "Brit. Med. Jour.," July, 1896.

[2] Notkin, "Wien. klin. Woch.," Oct. 22, 1896.

This serves to explain the various names applied to it in different countries.

Etiology.—Graves' disease belongs to the *reproductive period* of life, being rare before puberty and after the menopause. Barret[1] was able to collect only 42 cases in children below the age of fifteen, the youngest being four and a half. It affects *females* five or six times as frequently as males. A neuropathic *heredity* is commonly encountered. Pubescence in girls, menstrual difficulties, chlorosis, and all debilitating conditions act as predisposants. In exceptional instances Graves' disease seems to be due to certain intranasal and intra-abdominal conditions. As *exciting causes* may be mentioned emotional and mental *shocks*, especially profound and protracted anxiety and *grief*, but frequently cases attributed to such causes can be traced back of them. In such instances the mental strain may serve to precipitate the more prominent symptoms of the disease. *Pregnancy* may seem to excite the disease. Occurring during its course, the disease is sometimes apparently modified for the better, but, on the other hand, may be aggravated. After delivery there is frequently distinct improvement. Graves' disease is often associated with other neuroses, such as chorea, hysteria, and epilepsy. It is observed sometimes with tabes, and the mental disturbance not infrequently reaches into the field of insanity. A family type[2] is sometimes encountered, affecting several or all offspring of parents who may show no abnormality. A special but rare association of Graves' disease is with tetany, scleroderma, myxedema, and acromegaly. All of which are closely allied through relation to the thyroid body.

Numerous theories as to its *pathogenesis* have been entertained. Graves considered it a disease of the heart. Marshall, Taylor, and Piorry attributed it to mechanical compression of the cervical vessels and nerves. After the experiments of Claude Bernard upon the cervical sympathetic, lesions of this portion of the nervous apparatus were believed to be at the bottom of exophthalmic goiter. Following Charcot, the disease was by many considered a pure neurosis similar to hysteria. At present there are two general theories in the field. One incriminates the medulla, the other, the thyroid. Pointing to the *bulbar origin* of the disease is the association of cardiac, vasomotor, secretory, and thermic disturbances, to which, in various rare cases, palsies of cranial nerves, producing ophthalmoplegia, facial palsy, trigeminal neuralgia, and auditory disturbance are added. In certain cases bulbar hemorrhage, atrophy of the restiform bodies, and degeneration of the solitary bundles have been found, and the association with tabes argues the same cause. Felehne and Dardufi, by experimental lesions of the restiform bodies, have produced the major symptoms of exophthalmic goiter. As a rule, however, the bulb is not structurally affected, and the diverse conditions cited are as likely to be effect as cause.

Following Johnston there are many who believe that overactivity of the thyroid gland, causing a *hyperthyroidation*, explains the genesis of exophthalmic goiter. The principal facts supporting this point of view are: (1) The usual changes and enlargement of the thyroid; (2) the improvement and cures resulting upon removal of a portion of the gland; (3) the symptoms of exophthalmic goiter induced by overdoses of thyroids in myxedematous patients and normal individuals; (4) the cases in which exophthalmic goiter has eventuated in myxedema through degenerative changes in the gland, and (5) the almost absolute contrast between myxedema and Graves' disease.

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[1] "Jour. de Méd.," July 10, 1902.

[2] Brower, "Chicago Med. Rec.," 1898; Holmes, "Phila. Med. Jour.," June 11, 1898.

Horsley, who has given most constant and careful attention to this subject for many years, and to whose investigation we owe much of our present knowledge regarding the functions of the thyroid body, insists upon the changes in the gland and its secretion. He declares that "exophthalmic goiter in its various degrees results from perversion of the function of the thyroid gland."[1] It has been found by many observers that the use of thyroids in Graves' disease often intensifies all the symptoms, but there are certain cases in which they seem to cause improvement. Is it not reasonable to suppose that when the gland is simply or mainly overacting their administration increases the hyperthyroidation, but may benefit the cases that by perversion of thyroid function are not supplied with a normal secretion? Cunningham,[2] on experimental grounds, reaches with Gley the conclusion that the majority of symptoms in Graves' disease may be plausibly explained by the hypothesis of deficient thyroid activity. The question of the relation of thyroid action to the functions of the nervous system has been already outlined in the description of myxedema (see p. 499).

Accepting the thyroid theory, we are still confronted with the initial question as to why the thyroid function is primarily disturbed. Some have attributed this to infection setting up a thyroiditis, and point to the frequent history of antecedent infectious fevers, etc. Others accuse the neuropathic heredity or constitution. The real cause still escapes us. The intimate relation of thyroid and uterine functions (see p. 499) and the usual occurrence of exophthalmic goiter during reproductive life are of interest.

Morbid Anatomy.—The autopsical findings in exophthalmic goiter are inconstant and variable. The *heart* is often dilated; the sometimes thickened walls may present fatty degeneration. The valves are diseased only as accidental concomitants. The *thyroid body* may present any goitrous variation from simple congestion to the most destructive lesions. Usually the lobes are unequally enlarged, firm to the touch, and present a reddish, pulpy aspect. Colloid degeneration in places is not uncommon and may result in cystic formation. The vessels are thickened, dilated, and, in chronic cases, atheromatous. There is a proliferation of connective tissue throughout the gland which may reach a sclerotic degree. The acini of the gland become dilated; the colloid material disappears, leaving a granular débris; the secretion becomes thin and watery, the epithelium more or less disintegrated. The *thymus* is often persistent and enlarged, showing increased vascularity and an attempt at compensatory or perhaps at antagonistic activity. The *orbital cavity* is often normal, but as frequently presents some fatty proliferation, and almost always the evidence of a continuous retrobulbar congestion.

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[1] "Brit. Med. Jour.," Dec. 5, 1896. [2] "Jour. of Experimental Med.," 1898.

On the part of the *nervous system* the alterations in the cervical sympathetic ganglia, described in early accounts, are not found. Changes attributable only to the cachectic state are detected. In the bulb and spinal cord vascular degeneration has been encountered, with occasional small hemorrhages and dilatations. Atrophy of the restiform bodies and of the solitary bundles has been noted once or twice, as has atrophy of the ascending root of the trifacial. The associated lesions of tabes are more frequent.

The *muscles*, according to Askanzy,[1] show fatty infiltration, usually distinct to the naked eye. The fat globules are seen microscopically in long rows in the muscle-fibers, which also show increased, sometimes degenerated nuclei.

Symptoms.—The *onset* of exophthalmic goiter is frequently insidious, and the patient can scarcely say when it began. In other instances the major symptoms appear within a few days or even within a few hours, following some inciting shock, but it is always allowable to suppose that unobserved minor manifestations had been previously present. Such fact can frequently be elicited by judicious inquiry. Cardiac palpitation, nervousness, irritability, and unaccountable fatigue are often felt for months before the more prominent symptoms are declared.

Cardiovascular Features.—The *cardiac disturbance* usually is the first of the triad of cardinal symptoms to appear. The pulse becomes rapid, and ordinarily reaches 120 a minute, unless the patient is reclining and at perfect rest, when it may drop to 90, but never reaches normal during the activity of the disease. A rate of 150 or even 200 is not very rare. Of the three major symptoms a rapid pulse may alone exist, and, taken with several other and minor symptoms, may suffice for a diagnosis. It is the only essential symptom. Ordinarily regular, the rhythm of the heart-beat in some cases is greatly disturbed. The heart acts in the most tumultuous, irregular manner, and its incompetence is shown by vertigo, cyanosis, and precordial distress. Palpitation and cardiac throbbing are experienced by most patients, and may be occasioned by the slightest physical or mental distress. At such times the breast, neck, and face are frequently suffused, and hot waves, with violent blushes, may sweep over the upper part of the chest, neck, and face. At first, and sometimes throughout the disease, the stethoscope reveals nothing but the tachycardia and violent systole. Functional systolic bruits and anemic murmurs are not infrequent. Organic valvular disease is practically an accidental complication. An apparent hypertrophy is really due to dilatation and to the enlarged area over which the apex-beat extends by its increased violence. In late cachectic stages the dilatation, with degeneration in the heart-muscle, becomes physically apparent and threateningly prominent. Grocco[2] finds that dilatation is always present during attacks of tachycardia, that there is an intimate relation between the amount of dilatation and the general asthenia, and that intense but transitory changes in the sounds and shape of the heart are marked features of the disease.

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[1] "Deutsch. Arch. f. klin. Med.," Band 61.

[2] "Riv. crit. di Clin. Med ," Jan. 2, 1902.

The entire circulatory system is affected. This is most manifest in the aortic branches, especially in the cervical arteries, and is seen in the temporal and retinal arteries and veins. It is less well marked in the extremities, but Gerhardt[1] has noted it in the palmar arches and in the crurals. He also calls attention to the presence of capillary pulsation in the spleen and liver and kidneys. In cases of long standing, vascular dilatation is produced and the veins themselves become fibrous and arterialized.

The goiter may appear at any period of the disease or may never develop. In some instances it is of insidious growth, and the patient's attention is only incidentally called to it by tightness of the ordinary neckwear. In other instances it is of sudden development, or may advance in size at intervals, or advance and recede repeatedly. In any case it rarely attains very large proportions, and practically never interferes mechanically with respiration. It is usually asymmetrical, the right lobe commonly being most affected. The swelling may be confined to one lobe, usually the right, or it may involve the isthmus alone. The tumor usually offers considerable resistance to palpation, and a pulsatile thrill is frequently to be detected. In some instances nodules, indicative of parenchymatous changes, can be made out. Auscultation over the enlarged thyroid readily detects in most cases a bruit synchronous with the pulse, and the tumor can often be seen to expand with every systolic impulse. By manual compression it may often be greatly reduced in size for the moment, and it has a tendency to fluctuate in volume during the progress of the disease, increasing in size upon the occasion of any physical or mental excitement. In late cases, through interstitial thickening or cystic degeneration, it may give a sclerous or fluctuant feeling to the touch, and does not then recede upon the cessation of the malady. It may even become markedly atrophic, and then myxedema gradually develops.

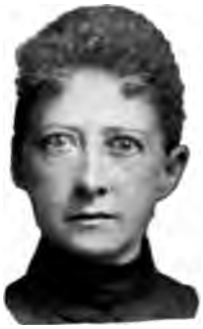
Fig. 221.—Facies in Graves' disease.

Ocular Conditions.—With the appearance of *exophthalmos*, which usually promptly follows the goitrous enlargement, the famous triad of symptoms is complete. It may appear before the goiter or in cases that never show thyroid enlargement. Both eyes commonly are affected, but at first usually in unequal degree, and exceptionally but one is prominent. This occurs usually in cases in which only one lobe of the thyroid is enlarged, and commonly on the same side.[2] The prominent eyeballs with wide-open lids give an expression of excited fierceness and fright strangely mingled, and quite disconcerting to strangers. The ocular protrusion varies greatly in amount in different patients, but in extreme cases has caused actual dislocation of the eyeball. Unless it exceed a moderate amount it occasions no inconvenience and may even escape the patient's attention. In a more pronounced degree the exophthalmos induces some difficulty in ocular movements and causes ocular fatigue. When exophthalmos is well marked, a distinct bruit has been heard by placing a stethoscope on the closed lids.[1] The cornea may also become inflamed from inadequate protection, especially at night and in the wind. There is frequently an increased *lacrimation*, which may occur independently of the exophthalmos, and later the secretion of tears may be defective. *Conjunctivitis* and *keratitis* may arise as complications, and *perforation* by ulceration has been known.

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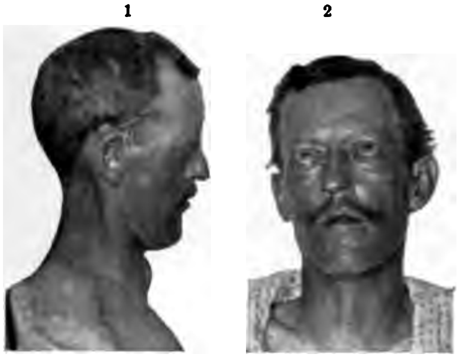
[1] "Centralblatt f. Chirurgie," Sept. 5, 1896.

[2] Fridenberg, "Med. Record," July 13, 1895.

The *lids* are usually retracted and often show peculiar and important motor difficulties. Winking is infrequent, but in occasional instances is rapidly repeated at momentary intervals. Von Graefe noted that in looking down the upper lid did not correspondingly follow the movement of the ball as in health. This sign is not related to the amount of exophthalmos, as it is sometimes found in normal individuals, and may be absent in marked cases of Graves' disease. Stellwag called attention to the usual widening of the palpebral fissure and the incomplete closure of the lids even when the patient thinks the eyes are firmly closed. In a few cases Joffroy has noted a similar inactivity of the lower lid and of the frontalis in looking upward, voluntary control of the latter muscle remaining perfect. The outline of the palpebral fissure is often more or less angular, losing its smooth curves. Möbius has called attention to a difficulty of convergence that is commonly present. In exceptional cases an external *ophthalmoplegia* has been seen. Paresis of the frontalis or of the entire facial nerve or involvement of the sensory and motor portions of the trifacial has been noted.



Figs. 222 and 223.—Exophthalmic goiter 1, Shows exophthalmos and the thyroid tumor; 2, the peculiar outline of the palpebral opening and the failure of the brows and lower lids to ascend in looking upward.

Vision is usually intact and the *pupils* are normal, but myopia may develop on extreme pressure from exophthalmos. The eye-grounds show no abnormality aside from retinal congestion and dilated, sometimes pulsating, vessels. Photophobia and brilliant muscæ are attributable to the retinal congestion, and hallucinations of sight may have the same origin.

Motor Conditions.—Besides the motor disturbances of the eye and its appendages, previously indicated, the entire musculature is affected with such marked weakness that Charcot was led to describe a Basedowian paraplegia. *Muscular asthenia* is often an early symptom. The reflexes may be diminished and the legs may suddenly give way, causing the patients to fall heavily. Of similar origin are the shallow respiratory excursions and diminished expansion of the chest. Cramps, contractures, fleeting tetanoid conditions, and even epileptiform crises have been noted. The occasional association of chorea, epilepsy, and hysteria has already been mentioned, and must not be confounded with motor disturbances due to Graves' disease.

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[1] Sanger Brown, personal communication.

An almost constant symptom in Graves' disease is a *fine tremor* which is as significant as any one of the cardinal triad. It is of variable intensity, may appear only at intervals, and be limited to the head and upper extremities. By placing the finger-tips on the head when the patient is sitting or standing, the examiner will be aware of a vibration, and this may be demonstrated by placing a long feather in the patient's hat. It usually is easily seen in the extended hands, especially if the fingers be spread widely, or it may be then felt by grasping the patient's wrist. In some cases the entire trunk is affected, and in standing, the tremor may be present in the lower extremities. It is sometimes distressing to the patient, but often occasions no complaint, and must be intelligently sought. The rapidity of the tremor is from eight to ten oscillations a second.

Secretory and Vasomotor Features.—With the hot waves and morbid blushing that have already been noted there is often profuse *perspiration,* which may be widely distributed, limited to one side of the body, or most abundant on the hands and feet. In some cases it necessitates frequent changes of underclothing. Usually it is attended by a feeling of almost *unbearable heat,* and these patients often seek cool rooms even in winter and find all bed-covering intolerable. The bodily *temperature* may be quite normal, but in rare instances an elevation of from one to five degrees has been noted, and in the terminal cachexia of fatal cases hyperpyrexia is commonly encountered. *Hemorrhages* from the uterus, nose, gums, and lips are not infrequent.[1] *Polyuria,* especially periodical polyuria, is a common phenomenon, and *glycosuria* is sometimes encountered. *Albuminuria,* however, is the rule at some period of the disease. It is usually intermittent and appears when the other manifestations of the disease are exaggerated, as if due to the hypervascularity of the medulla. *Edema* is common, but peculiar and usually circumscribed; it may affect the upper eyelids and has a preference for the outer aspect of the legs, the thighs, and the abdomen. The infiltration is not boggy and does not pit on pressure, but reminds one of the resiliency of myxedematous swelling. In rare instances it may become generalized and does so in those cases that eventually develop myxedema.

Mental Disturbances.—From the beginning, and often for a long period antecedent to the appearance of cardiac symptoms, the subjects of Graves' disease present a considerable *mental erethism.* There is an indefinable and tormenting agitation, marked by mental and motor restlessness and an imperative and impulsive tendency to be doing. Their emotions are too readily excited, and they are unusually impressionable and irritable, reacting in an exaggerated manner to all the incidents of daily life. In more pronounced cases they become voluble and manifest the greatest mobility of ideas, without persistent concentration or logical order. Their affections are likely to undergo modifications, and they become irascible, fault-finding, inconsiderate, ungrateful, and hard to live with. In some instances this disturbance of mentation carries them over the border into active mania, marked, perchance, by delusions of fear, due to the cardiac symptoms or the sensations of heat. Insomnia is often added, and the fitful sleep is disturbed by horrifying dreams that are likely to be projected into the waking moments and woven into delusions which are usually unsystematized, and constantly changing, furnishing the analogue of the motor restlessness. Hallucinations of sight and hearing are not uncommon.

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[1] S. Popoff, "Neurol. Centralbl.," April, 1900.

The mental perturbation only rarely reaches the degree of actual mania, and then is, perhaps, equally dependent upon numerous other causes acting in a neurotic individual. But a condition of *abnormal mental stimulation* is characteristic of the malady, and as important an index as any of the cardinal triad.

The skin is often marked by *abnormal pigmentation*, which may be distributed in irregular plaques over the body, face, and limbs, or in rare instances produces zebra-like markings on the trunk. In certain cases it much resembles the bronzing of Addison's disease, but usually spares the mucous surfaces, though not always. Vitiligo, scleroderma, and various cutaneous eruptions are sometimes encountered, and the hair may become thin. The sensation of heat is usually associated with a *diminished resistance to electricity*, which may be reduced to a third or a fifth of the normal. This is possibly related to the excessive perspiration, but does not obtain in other diseases in which sweating is equally great.

Digestive disturbances are numerous and not marked by anatomical lesions. Anorexia, voracious appetite, vomiting, diarrhea, dysentery, and icterus may be encountered in different patients, or many of them at various times in the same patient. The *diarrheas* are particularly important, as they rapidly reduce the patient's strength and tend to hasten the appearance of cachexia and a fatal termination by exhaustion. The frequent painless stools are made up of undigested food and an abundance of watery mucus, sometimes liberally streaked with blood. The defects in nutrition are always pronounced and a *loss of weight* from the first is the almost exceptionless rule. It may occur intermittently, the patient losing eight or ten pounds in a week and gradually regaining it. Huchard[1] is inclined to look upon such loss of weight, and especially upon its intermittent occurrence, as of diagnostic importance. It may occur independently of the diarrhea, sweating, polyuria, or any other physical drain, and in the face of an active bulimia.

Respiratory disturbances are not uncommon. They are frequently secondary to the cardiac disorder. Dyspnea, asthmatic attacks, pulmonary congestions, bronchitis, and a persistent cough, without stethoscopic symptoms, may be presented. The dyspnea is the same as that in myxedema, and appears to be due to the athyroidal condition (Horsley). Shallowness of respiration and inefficient thoracic expansion are related and proportionate to the muscular asthenia.

The *genital functions* are usually modified. Increased sexual appetite has been noted in men and women in the early periods of the disease. Later, as general asthenia develops, it is correspondingly reduced. Amenorrhea is present, or the tendency is well marked, and leukorrheas are abundant.

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[1] "Jour. de Méd.," Feb. 10, 1896.

The following table of symptoms and their relative frequency is based on Paessler's statistics,[1] comprising fifty-eight cases, of which forty-two were women. It must be borne in mind that nearly all the symptoms of Graves' disease are intermittent, and any tabulation, to be satisfactory, should cover the entire course of the disease and embrace a very large number of cases:

TABLE SHOWING RELATIVE FREQUENCY OF SYMPTOMS IN GRAVES' DISEASE, BASED ON FIFTY-EIGHT OBSERVATIONS.

Neuropathic family history	29	Sensations of warmth	24
Tachycardia	57	Vertigo	23
Nervous irritability and restlessness	57	Insomnia	21
Characteristic tremor	52	Stellwag's symptom	20
Palpitation and vascular disturbances	50	Tachycardia, goiter, exophthalmos, and tremor combined	18
Goiter	48	Polydipsia	17
Increased perspiration	34	Cardiac dilatation	15
Anemia	34	Irritable cough	10
Severe headaches	32	Graefe's symptom	9
Exophthalmos	28	Möbius' symptom	9
Severe diarrhea	26	Valvular heart disease	5

Course and Progress.—The great diversity encountered among cases of Graves' disease makes it difficult to outline its clinical course. It may be of apparently sudden or of insidious onset, and may show any combination of the long list of symptoms that have been detailed above. It may run its course to a favorable termination in three months or may take six years, or continue throughout or terminate life. The great majority of cases are of protracted duration. Early recoveries and fatalities are alike exceptional, and when all other symptoms have subsided, promi-

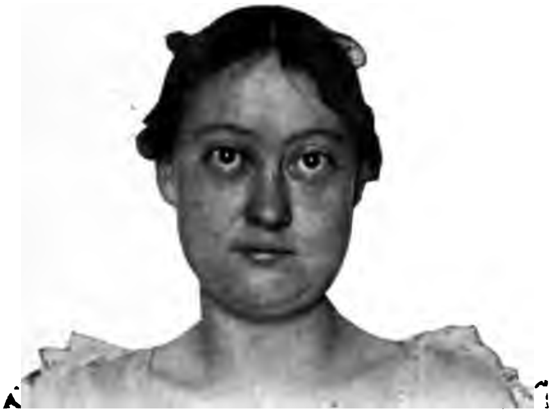

Fig. 224.—Partial recovery. Some exophthalmos, pigmentation of skin, and goitrous thickening remain.

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[1] "Deut. Zeit. f. Nervenheilk.," Bd. vi, S. 21.

nent eyes and a moderate goiter may remain to mark the passage of the disease. The appearance of active diarrhea, of polyuria, intense albuminuria, and rapid wasting darkens the prognosis. Extreme asystole, great cardiac dilatation, and a failing circulation have the same import. Maniacal disturbance is not of itself of bad prognosis, which depends rather on failing bodily conditions. The cardiac symptoms are usually the first to appear, and afford the best criterion as to the progress of the disease. Their improvement is the only unqualified sign of gain. A state of nervous susceptibility is usually left after recovery that may be compromised by the slightest shock.

Diagnosis.—In the presence of the Basedowian triad diagnosis can not fail, but in the abortive forms it requires a careful and thorough investigation and an experienced judgment to recognize the disease. Tachycardia, with irritability, sweats, insomnia, emaciation, and perhaps a slight rise in temperature may mimic *phthisis* very closely, and if an irritable cough is added, the mask is almost impenetrable, especially as some pulmonary dullness, due to the congestive condition, may be present. The mental attitude is of some assistance, and should the tremor be observed or some slight thyroid turgescence be made out, the diagnosis may be effected. Some of these *formes frustes* are put down as *neurasthenia*, in view of the asthenic condition so commonly present. It requires the presence of eye, thyroid, cardiac, or tremor symptoms to make the diagnosis positive. Every case of Graves' disease is at the same time myasthenic, and may also be neurasthenic.

Treatment.—In the management of this disease, whatever line of treatment may be added, it is of the utmost importance to secure *rest* and *quiet*. With complete rest, as by the Weir Mitchell course, some cases make immediate and substantial progress. If this is not available, it should be approached as nearly as the circumstances of the patient permit. In the asthenic, irritable state, a conservation of energy is strongly indicated. *Digitalis*, *strophanthus*, and *strychnin* have a favorable action on the heart in some cases, and strychnin in full doses is occasionally of benefit to the general nervous condition. Sedatives such as *bromids* and *belladonna* have not yielded the writer good results in any instance, but chloral is often valuable for the insomnia. *Iron* must be used with caution even to combat the anemia so commonly present, as it frequently disturbs the intestinal tract and augments the vascular storms. *Hydrotherapy* and *massage* are of benefit in some cases. *Electricity* locally has strong advocates, some favoring faradism, others galvanism. A moderate faradic current through the root of the neck, causing all the anterior cervical muscles to contract about the thyroid, mechanically diminishes it in size. This may last for a few minutes or for a few hours after the current is stopped if the patient is not excited in any way. An uninterrupted galvanic current may produce a similar result, and passed through the temples occasionally causes a recession of the eyeballs, but the effect is very fleeting and does not influence the tachycardia. Electricity should be used daily, or several times a day. Mechanical compression of the thyroid by bandaging is insupportable.

Injections of cicatrizing agents, such as tincture of iodin, into the substance of the gland, at one time much in vogue, are now generally abandoned, as they are of doubtful value and are attended by considerable danger to life. In long-standing cases *ablation* of a portion of the gland has produced good results in the hands of various operators, but it has a considerable mortality—about twelve per cent. Schulz[1] reports fourteen cases with twelve cures, but Sänger [2] cites a case in which all symptoms were aggravated. Cystic portions may be removed, but caution must be exercised to leave a fair amount of normal gland. The same effect is obtained by exposing the gland by a median incision and securing it in the wound (the *exothyropexy* of Jaboulay), but even this comparatively simple operation has been followed by death. Thus operated, the thyroid shrinks in size and the symptoms decrease. The wound is closed at a subsequent period. Jonnesco[3] and others have had improvement from bilateral ablation of the cervical sympathetic. The operation is a serious one and not without bad results.[4]

Thyroids have been used. Ordinarily, they intensify the symptoms. In a minority of instances the goiter is reduced in size quite promptly, but the pulse is usually not improved, and the nervousness and tremor are generally accentuated. They may be carefully tried experimentally. A number of cases have been benefited by the use of thymus feeding. Owen [5] has collected about twenty cases in which its effect was mainly beneficial. Mackenzie,[6] in an equal number of cases, found it of little or no value. Owen called attention to the probable physiological antagonism between thymus and thyroid action. The persistence or reappearance of the thymus in these cases, often noted, among others by Hektoen,[7] may have the same significance. Some encouraging results have been obtained by feeding patients on the milk of thyroidectomized goats or using a serum obtained from the blood of thyroidectomized animals. Several hemic products from such animals are being put upon the market, but their value is as yet undetermined.

The numerous *complications* must be met as they arise. Atropin for the sweats, bismuth and opium for the diarrhea, careful dieting for the gastric and intestinal troubles, and a general tonic and upbuilding régime. Thompson, of New York, insists on a milk diet.

SCLERODERMA.

Scleroderma is a trophoneurosis presenting an induration and atrophy of the skin, or atrophy without preceding induration. It occurs (1) in a generalized form, but affecting the extremities and face principally, and (2) in a circumscribed form, marked by isolated plaques and stripes. These may be confined to one side or be roughly symmetrical in distribution, and are often limited to the cutaneous extent of certain nerve-trunks or branches. This has led Jonathan Hutchinson [8] to use the term herpetiform morphea. The disease is also known as the hide-bound disease, scleroma adultorum, sclerosis, chorionitis, morphea, and Addison's keloid. When the fingers are affected, the French employ the term sclerodactylie. It is a rare disease, but not so uncommon as most writers insist, and if generally recognized would soon present a generous material.

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[1] "Berliner Klinik," June, 1897.

[2] "Munch. med. Wochens.," April 6, 1897.

[3] "Centralbl. f. Chir.," Jan. 16, 1897.

[4] Aschard, "Rev. de Neurol.," Aug., 1900.

[5] "British Med. Jour.," Oct. 10, 1896.

[6] "American Journal," Feb., 1897.

[7] "International Med. Mag.," Sept., 1895.

[8] "Brit. Med. Jour.," June 1, 1895.

Etiology.—The female sex furnishes about three-fourths of all cases. Generalized scleroderma is most common in adults, but may be encountered between ten and twenty years of age, and the discrete form begins perhaps more commonly before twenty-five than later in life. It has been attributed to various infectious fevers, to traumatism, exposure to cold, rheumatism, and to pretty much every incident of human life. A neuropathic constitution is of such striking frequency in these cases that it can not be overlooked. Spinal cord and cerebral lesions are sometimes associated, and in the generalized form Raynaud's disease is a frequent concomitant. Insanity is sometimes present, especially melancholia.

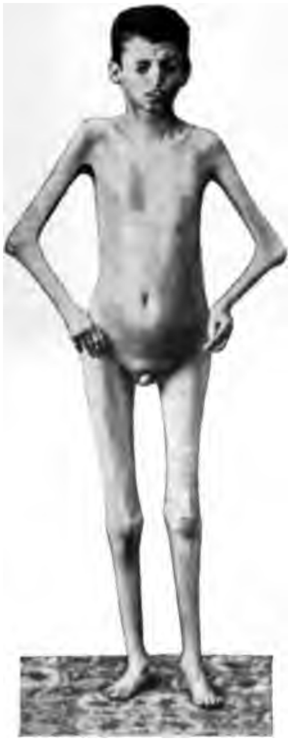
Fig. 225.—Generalized scleroderma, showing facies, sclerodactylie and hide-bound state (Grasset).

Symptoms.—In the generalized form of scleroderma at first there is usually some pain and sensitiveness in the part affected. This is followed by an induration and thickening that may be red and suffused by vascular stasis and present blebs and bullæ. Factitious urticaria and dermographia are commonly easily demonstrated.[1] On the broad surfaces of the trunk the skin feels stiffened and brawny. It is found to be bound down to the underlying parts, giving rise to the hide-bound descriptive title. Atrophy occurs later. All the dermal structures become thinned and present a cicatricial appearance, and may be glistening white. The hands and face are especially affected. The fingers are reduced to their slightest proportions, and the bones, covered by the atrophic, glistening skin, which fixes the joints and limits motion, are lessened in size. Raynaud's disorder is often present. Over the facial bones the parchment-like integuments are tightly drawn in a motionless, unwrinkled mask. The thinned lids can scarcely be closed over the sunken eyeballs, which consequently appear unduly large. The nose is thin and looks ready to split its way through the skin. The lips are papery and often retracted, exposing the teeth. The shortening of the skin from the jaw to the clavicles may even interfere with movement and with deglutition. Every bony prominence, as about the orbits, cheeks, and chin, is sharply defined. The epithelial structures are everywhere reduced, and nails and hairs are of defective growth. Sensibility is not much disturbed, as a rule, but hypersensitiveness may be encountered.

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[1] Launois, "Nouv. Icon. de la Salpêt.," 1900.

In the discrete form a local thickening may gradually invade the surrounding parts, but is limited by rather sharp borders and may be attended by herpetic eruptions. Later it becomes atrophic. In other cases a white atrophic spot appears and gradually extends over the course of the supplying nerve. It may thus produce zoster-like bands on the trunk and stripes down the limbs. The most common locations are the face, chest, and lower extremities. The outlines of these morpheic tracts and plaques sometimes forcibly remind one of the cutaneous areas of the spinal segments.

Up to the time atrophic changes occur, the disease may recede and the normal condition be spontaneously established. Several years are usually consumed in the development of the disease.

Anatomically, the disease is marked by a fibrosis attended or preceded by vascular changes. The nerves and all structures within the atrophic area equally show the fibrous proliferation.

Treatment.—Tonics, electricity, hydrotherapy, massage, and general reconstructive measures have been found of some benefit in a few cases. Of late, Lustgarten, Sachs, Bramwell, and others have reported great benefit from thyroid feeding, which, even in advanced cases, caused immediate and gratifying changes for the better. In recent cases it appears likely to produce a cure. Whether a relapse will follow discontinuance of the remedy is not now certain.

RAYNAUD'S DISEASE.

Raynaud, in 1862, described a peculiar dry gangrene, especially in the extremities, which he attributed to a disturbance of the vasomotor apparatus. From present information the condition may better be considered as a generalized one, but with local exaggeration. Naturally, the circulatory disturbance is most apparent in the extremities, as in the fingers, toes, nose, and ears. The arterioles and venules are spastically contracted during the attack. Raynaud described three stages, which he strikingly named local syncope, local asphyxia, and local death. The condition is usually symmetrical, may recede at any stage short of gangrene, and usually appears many times in succession in mild degree before inducing mummification.

Etiology.—Females furnish twice as many cases as males and no age is exempt, though most cases occur between twenty and forty-five. All varieties of anemia are strongly predisposing factors, and a neuropathic makeup is almost invariably present. Other nervous diseases are commonly associated, such as hysteria, epilepsy, tabes, neurasthenia, myelitis, and insanity, especially acute mania. Urticaria, telangiectasis, angioneurotic edema, and scleroderma may be combined in the history or present in the patient. Urticaria and local asphyxia may alternate in the same patient. Heredity is apparent in ten per cent. of the cases showing the same disorder in blood relatives. Nekam[1] reports seven

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[1] "Neurolog. Centralbl.," Oct., 1904.

children of one mother, who had been twice married, affected with this vascular defect. Any occupation attended by exposure to cold and wet may play a part in the causation. Any sudden demand upon the physical strength or powers of resistance may induce the attack. Exposure to cold is the most common immediate cause, but fright, grief, fatigue, trauma, influenza, malaria, and acute infections may induce it. Some consider the ultimate cause of the angiospasm to be an auto-intoxication. It is often associated with scleroderma.

Symptoms.—The local symptoms first attract attention. The fingers and toes, less frequently the ears, nose, and lips, or a single finger, appear pale, waxy, bloodless, and glossy. There is usually a feeling of tingling, numbness, and loss of sensibility, as in a finger compressed by an elastic bandage. A needle-prick draws no blood and the finger looks dead; hence the term *digiti mortui.* After lasting a few minutes to several hours, this condition, often attended by chilliness, nausea, and general discomfort, may pass off or develop the second stage of local asphyxia. The affected part becomes cyanotic, blue-black, and the anemic pressure-trace disappears very slowly. Less commonly the fingers in this stage may puff up, present a vivid red color, be extremely hot, and covered with perspiration. There is now usually more or less neuralgic pain proportional to the cyanosis. Both hands may be simultaneously affected or one may precede the other. One finger may, in the second stage, present the extreme blue-cold cyanosis, its neighbor the turgid, hot condition or the white, syncopic stage. If the second stage persists long enough, several hours at least, small blebs appear, raising the epidermis especially from the pads at the finger-tips, ulceration follows, and dry gangrene may mummify and destroy the terminal phalanges or entire fingers. Small necrotic patches may form, and, healing slowly, leave cicatrices to mark the attack. The amount of mutilation, fortunately, is often insignificant in relation to the extent of cyanotic tissue. A hand or foot that appears doomed may only lose a few phalanges. The disease may appear in other portions of the body, as in patches over the deltoids, inner aspect of the calves, the heels, maleoli, nates, cheeks, and on the abdomen, rarely, however, going on to gangrene. The genitals and tongue are exceptionally attacked.

Constitutional symptoms rarely default. Intermittent hemoglobinuria, uremia [1] with diminished urea and a lessened quantity of urine, and uremic convulsions are frequently noted. Attacks of colic have been met with during the angiospastic attack. There is no fever at any period except from coincident febrile disease. Cerebral disturbance is very common.[2] Irritability, depression, aphasia, unconsciousness, coma, convulsions, and mania have all been noted. The kidney symptoms and the brain disturbance are apparently due to the angiospasm, which further shows in the narrowed retinal artery, dimness of vision, occasionally occurring hebetude, tinnitus, ageusia, and iridoplegia.

Course and Prognosis.—The attacks are of variable duration and intensity. A few hours is usually sufficient for the local syncope and asphyxia to develop and subside, but ordinarily another exposure to cold or the incidence of any mental strain or physical fatigue occasions a return. When gangrenous changes have developed, several months are usually consumed in exfoliation and cicatrization, as the process is extremely slow and healing very tardy. In infants the disease is of rapid course and may terminate in death in a few days. In older patients recovery is the rule, but uremia may cause death.

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[1] Aitken, "Lancet," Sept. 26, 1896.

[2] Osler, "Amer. Jour. Med. Sciences," Nov., 1896.

Diagnosis.—The diagnosis in a typical case is not difficult. The causes of ordinary gangrene are lacking, but nephritis may be present in Raynaud's disease. Repeated symmetrical local syncope, followed by regional asphyxia and passing away in a few hours, can scarcely be mistaken. Several bouts of lesser degree usually precede the gangrenous variety, often appearing during the winter for years in succession. Hemoglobinuria or retention of urea greatly assists the diagnosis.

Treatment.—The principal indication is to improve the nutrition and reëstablish the general health. Locally, during the attack, the use of warm applications is indicated. The parts may be wrapped in cotton and the temperature properly maintained by artificial heat. Trinitrin and amyl nitrite sometimes give prompt relief. Morphin, hypodermatically, is sometimes required to relieve pain. The local use of the constant electric current has been much advocated, but the warm salt solutions, in which the extremities are directed to be immersed, probably are as active as the electricity. Cushing [1] strongly recommends the application of an elastic or Esmarch bandage to relieve the attack. When the bandage is removed, the parts rapidly fill with blood and the vascular spasm subsides. The treatment of the gangrene is surgical, but it is especially needful to wait for the demarcation line, as it often includes comparatively little of the threatened extremities.

ACROPARESTHESIA.

Schultze first, in 1890, used the term acroparesthesia to describe a condition which had previously been fully recognized by Nothnagel, Bernhardt, Putnam, and others. It occurs generally after middle life in hard-working women who have the hands constantly wet, as in washing and scrubbing. The paresthetic feeling is usually a tingling, crawling, or burning sensation, sometimes attended by decided pain and most marked night and morning and in winter. The hands are usually alone affected, but the feet may participate. The fingers may be rendered clumsy and awkward. The color of the skin is usually natural, but may be pale, whitened, or reddened. Sensibility is commonly objectively normal, but hyperesthesia and hypesthesia have been observed. Arteries, veins, and nerves are normal. There is no atrophy of muscles or integument, but occasionally a complaint of weakness. The disorder is not serious, but tends to chronicity and may last many years. Cassirer [2] notes only 12 men in 162 cases; and 106 out of 129 cases occurred between the ages of thirty and sixty. All occupations attended by exposure to cold and dampness are represented. The climacteric, alcoholism, influenza, parturition, and uterine extirpation are also noted as causal. The paresthesia is never limited to the distribution of a given nerve, but usually affects all the digits about equally, may extend to the elbows or even to the shoulders, and is bilateral as a general rule. The feet are also affected, but less frequently and less severely.

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[1] "Jour. Nerv. and Ment. Dis.," Nov., 1902.

[2] "Die Vasomotorisch-trophischen Neurosen," Berlin, 1901.

The **diagnosis** must exclude Raynaud's disease, multiple neuritis, and scleroderma.

The **prognosis** is not good as to early recovery, but favorable as to any serious resulting condition.

In **treatment** the faradic brush, the static spark, and local galvanism have been accredited with favorable action. General measures, tonics, hydrotherapy, and, most of all, avoidance of occupational causes are mainly to be relied upon. Collins found intestinal disturbances of some sort in 57 out of 100 cases.[1] All such conditions require unremitting attention.

INTERMITTENT LIMPING.

A condition first described by the French under the term *claudication intermittente*, has been designated *intermittent limping* by the English, *intermittirende Hinken* by the Germans, *angiosclerotic paroxysmal myasthenia* by Higier,[2] and *angina cruris* by Walton.[3] The condition is manifested in pains and paresthesia, generally in the feet and legs, intensified on attempts at walking, and in severe cases preventing the patient advancing more than a few steps at a time. Usually the feet are more comfortable in a dependent position, and in some cases the patient can only obtain sleep by allowing the feet to hang over the edge of the bed or by sleeping in a sitting posture. A recurring lameness has been long known to veterinary medicine and is found to be due to disease of the aorta, generally an aneurism, interfering with circulation in the hind extremities. In the human race it is always associated with, or perhaps one should say is the manifestation of inefficient circulation in the legs and feet. In the majority of instances no pulse can be obtained in the dorsal artery of the foot, and in a large proportion of the cases the posterior tibial is also pulseless. The pain, which is the chief complaint, is manifested in three different conditions: (1) Pain upon walking; (2) permanent pain, frequently in the form of painful paresthesia while the patient is at rest; (3) the pain which initiates true gangrene.

Goldflam[4] in twenty-four cases found the disorder at twenty-five years of age in the youngest, but usually the condition develops in middle life and rarely after fifty. This author believes that the pathological condition is usually an endarteritis, although it is but seldom that any general arterial sclerosis is present. Erb has insisted upon an inherited disposition, and others contend that there is a congenitally defective arterial system. The abuse of tobacco or alcohol has been rather frequently noted in these cases, but the withdrawal of either or both toxic factors does not appear to better the condition. Syphilis was only found once in the twenty-four cases of Goldflam. The condition is usually symmetrical, and in a few instances the nerves of the part have been found histologically changed slightly, presenting those variations which are encountered in an arteriosclerosis. Massaut[1] has reported a case in which the upper extremities were similarly affected in a woman of twenty-four and no arterial pulse could be found in either arm. The painful paralysis of the upper extremity recurred upon any continuous use of the parts. In the majority of instances the feet are cold or blanched, but in some cases the parts are intensely reddened, suggesting an erythromelalgia, and in others the alternation between blanching and redness strongly simulates Raynaud's disease. It is not unlikely that the three conditions may be combined. Flat-foot has been observed in a large proportion of these cases.

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[1] "Med. Rec.," May 31, 1892.

[2] "Deut. Zeit. f. Nervenheilk.," July, 1901.

[3] "Boston Med. and Surg. Jour.," April 3, 1902.

[4] "Neurol. Centralbl.," Mar. 1, 1901.

Medicinal treatment seems to have little value. Even the alkaline iodids, which have such a well-founded reputation in chronic arterial changes, give little assistance. Warm baths, massage, electricity in the form of the galvanic current, withdrawal of tobacco and alcohol, rest and general physical improvement are the measures to be adopted. Gangrene when once it appears has a tendency to extend, and early amputation is generally recommended.

ANGIONEUROTIC EDEMA.

Angioneurotic edema, otherwise known as *acute circumscribed edema, acute non-inflammatory edema, giant urticaria, periodic swelling, giant swelling, Quincke's disease,* etc., was first definitely described by Milton, in 1876, as giant urticaria. Dinkelacker and Quincke, in 1882, gave prominence to it under the above title. In 1892 Collins was able to assemble seventy-five cases and made a critical digest of the literature. Since that time numerous instances have been reported all over the world. It is marked by acute circumscribed swellings of the subcutaneous or submucous tissues, often presents a direct heredity, usually appears in those of a neuropathic diathesis, is often periodic or recurrent, and is commonly attended by gastro-intestinal colics.

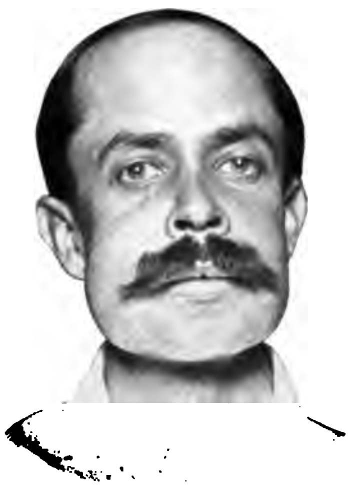

Fig. 226.—Angioneurotic edema involving the lower portion of the face in a very spare individual.

Etiology.—The most active *predisposants* are heredity and exhausting conditions. The neuropathic taint may show itself in antecedent or associated neuroses and psychoses in the family or in the patient, or by the stigmata of degeneracy. This, however, is far from being an absolute rule, and numerous cases give no clue whatever to the origin of the neurotic disturbance. Direct heredity is often marked, as in the series reported by Osler,[1] in which the disease numbered 20 cases, extending through five generations, and the series of Milroy,[2] showing six generations and 22 cases out of 97 individuals. Continued mental or physical exhaustion often prepares the field for the development of the vascular neurosis. The greatest number of cases appear early in adult life, from twenty to thirty-five years of age; but it has been noted in infancy, or in the marked hereditary cases it may be congenital, as in 20 out of 22 cases of Milroy. Both sexes are affected, but females somewhat more commonly than males. In several cases a decided gouty tendency was present. The urine showed a diminished quantity of urea, and the attacks were beneficially modified by reduction of nitrogenous diet.

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[1] "Ann. de la Soc. méd.-chir. d'Anvers," Mar., April, 1901.

As *exciting causes*, exposure to cold, intestinal disorders, puberty, the climacteric, masturbation, traumatism, fright, grief, and the action of toxic agents, such as tobacco, alcohol, and malaria, have been noted with considerable frequency. In some instances the malarial intoxication has caused quotidian or tertian attacks. In some they have recurred every seventh or twelfth day. Aside from such periodicity they have a tendency to develop during the latter half of the night, when the circulatory activity is at its minimum. They also show a seasonal increase in winter and summer, apparently the result of temperature changes and chilling of the skin, and sometimes they follow slight blows.

Symptoms.—The swellings of angioneurotic edema generally appear without warning, and reach their maximum in a few minutes or in an hour or two. The most usual locations are the face, lips, tongue, pharynx, genitals, and extremities. The hands are frequently affected. The buttocks, shins, or abdomen may be selected. The swelling is tense, sharply defined, not tender, does not pit on pressure, is whitish or pinkish, and rarely marked by purpuric discolorations. It lasts from a few hours to several days, and vanishes as rapidly as it appears, leaving no trace, or as it leaves one locality a similar swelling may show itself elsewhere without symmetrical or anatomical relation. Several swellings may appear at once. In amount, the swelling varies greatly, but may attain large proportions; hence the name giant swellings. Sometimes nodular swellings as large as hen's eggs are encountered. There is usually a subjective feeling of tension and stiffness in the parts, and some burning, prickling, or itching. If the skin is scratched, urticarial stripes and wheals usually appear, or they may attend the attack or alternate with it.

The tongue, larynx, pharynx, stomach, and intestines are sometimes affected by the swelling, and local discomfort and even serious danger to life may result. Dyspnea, difficulty of swallowing, intestinal and gastric colic are thus induced, and several cases have perished from the laryngeal occlusion. Gastro-intestinal symptoms are very common and appear in from one-half to one-third of the cases. A feeling of uneasiness in the epigastrium is followed by distention, nausea, and obstipation. Colic, cramps, profuse vomiting, and intense thirst ensue. The attack terminates with a colliquative diarrhea. There is often a great increase in the urinary excretion during the attack, and albuminuria and hemoglobinuria are not infrequently encountered. An effusion into the joints sometimes takes place. The attacks may come on at various intervals of days, weeks, or months, or with regularity, and ordinarily between the attacks the previous general condition of health is regained. A blow is sufficient to induce and locate a swelling in some cases, and there is a tendency for the swelling to recur constantly in the same locations. In some of the congenital cases the swelling is practically permanent, general as well as intestinal symptoms being absent. Occasionally there is an association of erythema and angioneurotic edema, the redness and swelling being of similar or dissimilar distribution.

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[1] "Amer. Jour. Med. Sciences," April, 1888.

[2] "New York Med. Jour.," Nov. 5, 1892.

Diagnosis.—The diagnosis can present little difficulty if one is on guard. We must differentiate the erythematous nodosities of rheumatism, the persistent blue and white edemata of hysteria, and the edemata of renal and cardiac origin.

Prognosis.—There is little danger unless the larynx be affected, and there is a general tendency for the attacks to cease in advanced years, though sometimes they last for life, or may reappear after a long interval. If traceable to inciting causes, the immediate prognosis is improved. If dependent upon toxic conditions due to alcohol, tobacco, malaria, lithemic, or gastro-intestinal infection, the proper intervention promises good results.

Treatment.—If the cause can be discovered and removed, the neurosis promptly yields. Otherwise reliance must be placed on those general measures which are fitted to combat the neuropathic constitution and build up the usually depraved systemic condition. The treatment of the attack is symptomatic. Morphin for gastro-intestinal crises, heat and compression to the swelling, and tracheotomy or intubation if respiratory failure is seriously threatened. Bloodgood[1] reports several cases of persisting angioneurotic erythema cured by deep incisions.

LOCALIZED HYPERTROPHIES.

Symmetrical Lipomatosis.—Occurring commonly in adults, and usually on a background of syphilis or alcoholism, are cases manifesting localized and symmetrical fatty deposits. These may be tender and the seat of spontaneous pain or comparatively insensitive, and the pain may be quite insignificant. Their favorite location is about the neck, the *diffuses Lipoma des Halses* of the Germans; over the body, as in the case of Hugièr, which presented twenty symmetrical pairs; in the axillæ and groins; or on the extremities, as in the case reported by Mathieu, one pair over the trochanters, one pair on the inner side of the knees. The *adiposis dolorosa* of Dercum, the cases of Peterson and Loveland, and the adenolipomatosis of Launois and Bensaude[2] (Fig. 228) appear to belong to the same group. Peterson, Collins, and others look upon the condition as due to a rudimentary polyneuritis, and the idea is supported by the common history of alcoholic addiction or luetic infection. In addition, sciatic neuritis, herpes zoster, optic-nerve symptoms, and other distinct indications of neuritic affection have been found in association. The local sensitiveness, pigmentary disturbances, and muscular flabbiness and weakness point the same way. In certain cases the reaction of degeneration and a wasting of the thenar muscles have been seen. Dercum[1] has finally demonstrated an interstitial neuritis. This has been confirmed by Burr,[2] and both found suggestive changes in the thyroid gland. A neuropathic tendency can frequently be traced. The condition has been mistaken for myxedema in instances marked by diffuse lipomatosis, but the hands, feet, and face are spared. It is of interest to remember that in five cases a diseased condition of the thyroid has been found post mortem.[3]

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[1] "Johns Hopkins Hosp. Bull.," May, 1903.

[2] "Nouv. Icon. de la Salpêt.," 1900.

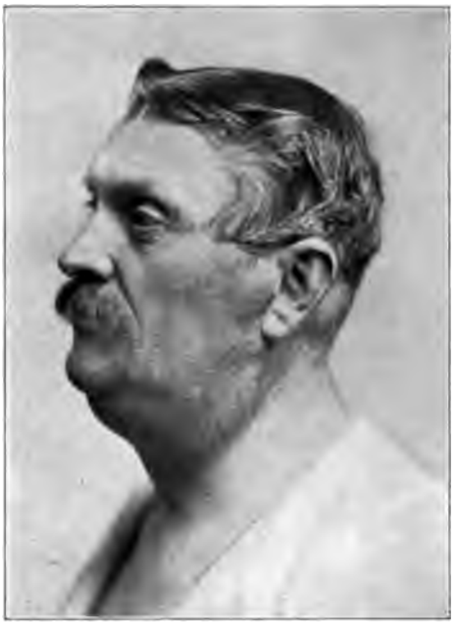

Fig. 227.—Symmetrical lipomata; three pairs, one over mastoids, one over maxillary articulations, one under angle of jaw (Beck).

The ill-defined fatty masses are commonly spread out at their borders into the surrounding tissue. They may, however, be globular or pyriform and pendent. Their common location is subdermal.

In slighter cases discontinuance of the use of alcohol or antiluetic treatment has caused the tumors to recede. In aggravated cases medical treatment has apparently accomplished little or nothing, but recourse to thyroid feeding promises more, in view of its effect on obesity and the, at least occasional, presence of disease of the thyroid in these cases. Surgical removal of the fatty tumors is sometimes indicated.

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[1] "Jour. Nerv. and Ment. Dis.," Aug., 1900.

[2] *Ibid.*, Oct., 1900.

[3] Dercum and McCarthy, "Amer. Jour. Med. Sciences," Dec., 1902.

Unsymmetrical Hypertrophies.—In rare instances, usually congenital, and frequently of neurotic ancestry, one side of the body or one extremity, or a portion of an extremity, as a hand or one or more digits, may be disproportionately large. The asymmetry usually increases with the child's growth. In still rarer cases it makes its appearance after birth, and may occur at any time up to maturity. The hypertrophy usually involves the affected portions *en masse*, so that the increase in length is proportionate to the breadth and thickness. The principal increase is usually in the adipocellular tissue, but the muscles may be hypertrophic and may show correspondingly increased strength.

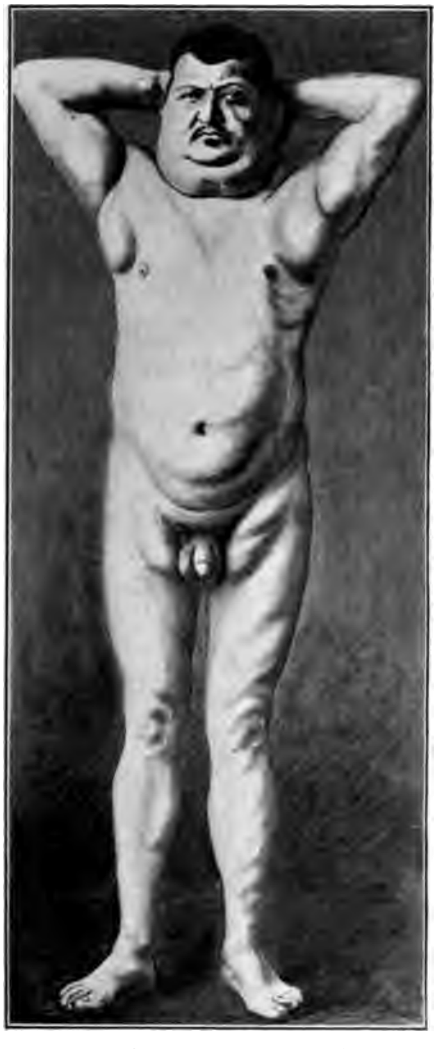
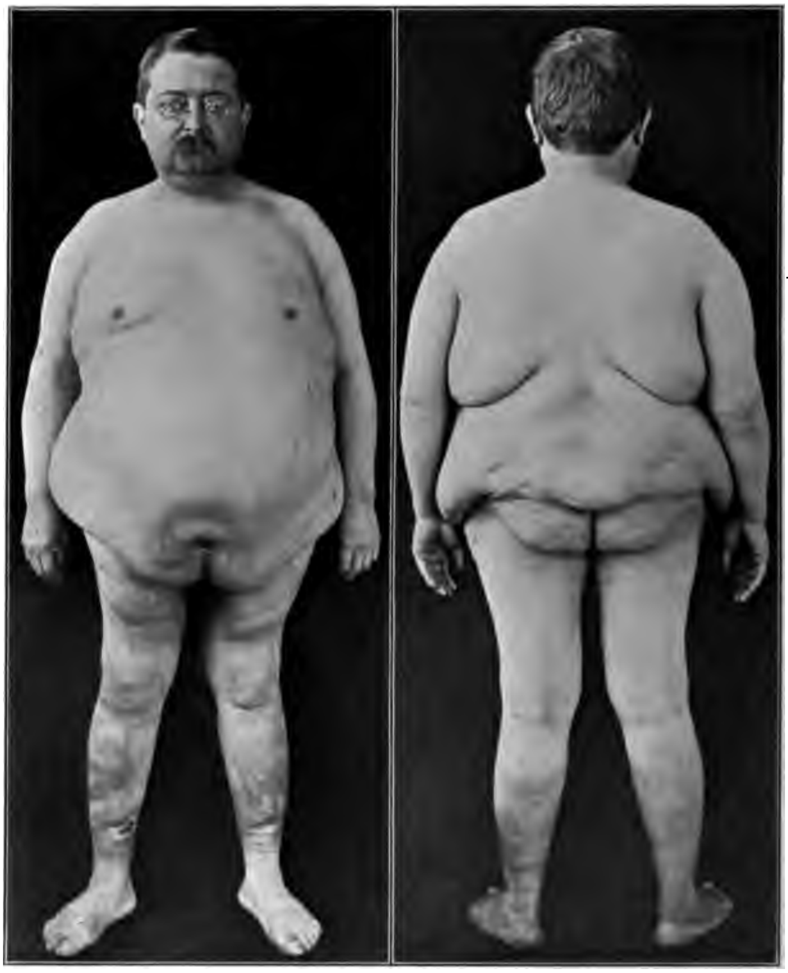


Fig. 228.—Symmetrical adenolipomatosis (Launois and Bensaude).

Usually, however, the muscles are defective. The bones are simply enlarged. Sometimes the hypertrophied parts are warmer than their normal fellows and may show increased perspiration and evidences of hyperemia. Occasionally there are pigmentary markings. Hemihypertrophy of the face may be encountered, and Friedreich reports a case presenting hypertrophied left face and arm and right leg. In rare instances such localized hypertrophies are seen in gigantism and acromegalia. Of their nature we know practically nothing. In some instances it has been attempted to check the overgrowth by compressing the arterial supply and by the injection of astringents, but no good seems to have resulted. Enlarged digits may be amputated.



Figs. 229, 230.—Adiposis dolorosa of the diffuse truncal form (Dercum). Fig. 229 shows the peculiar apron of fat and the small size of the hands. The posterior view shows the arrangement of fat in folds over the hips.

Chronic Hereditary Tropho-edema.—Henry Meige [1] describes a family in which edema affected eight members, both men and women, distributed through four generations. Four of these cases were observed, and present the same singular affection: namely, a chronic white, firm, and painless edema, appearing at the age of puberty, and affecting especially the feet and legs and sometimes the entire lower members,

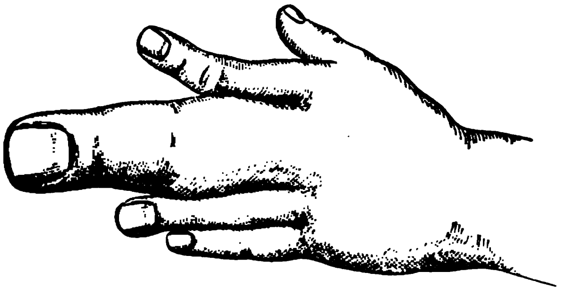


Fig. 231.—Macrodactyly: localized hypertrophy of a single finger (Ridlon).

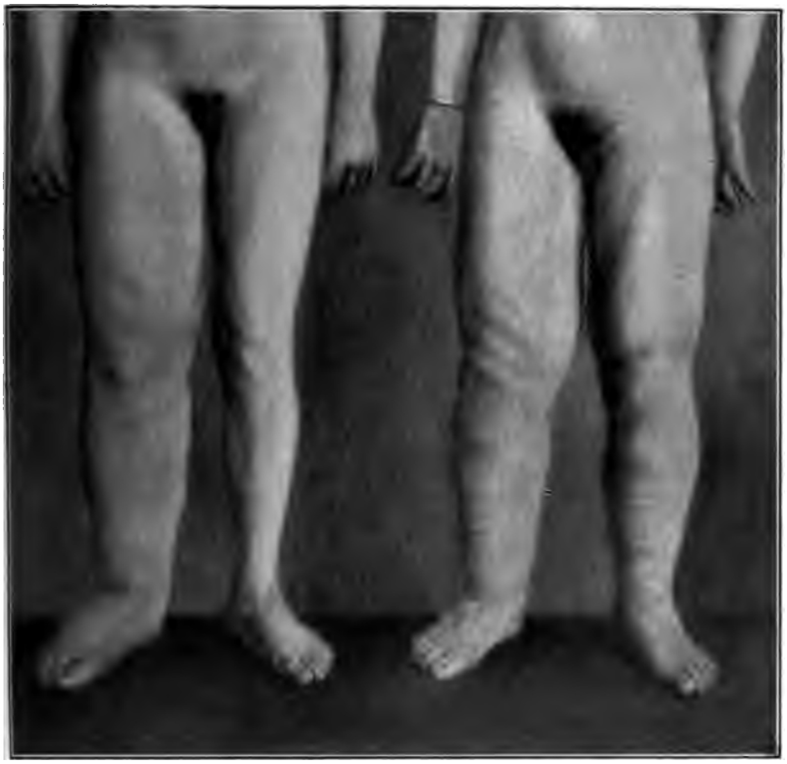


Fig. 232.—Chronic hereditary tropho-edema in sisters at seventeen and twenty-one years of age (Meige).

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[1] "Nouv. Icon. de la Salpêt.," Dec., 1899.

generally on both sides. He also refers to a remarkable family reported by Milroy,[1] in which in six generations there were twenty-two cases. Other similar family groups have been published, and A. Thomas thinks that one-sided hypertrophies of the body belong to the same category.

The condition in all known instances has caused but little inconvenience, has been attended by no suffering, has not shortened life, and has resisted all forms of treatment.

[1] "New York Medical Rec.," 1893.

CHAPTER II.

INFECTION NEUROSES.

THE diseases now temporarily grouped among the neuroses because the essential histological lesions of the nervous apparatus still escape us, but due to the action of infections, are *tetanus* and *hydrophobia, tetany* and *chorea*. In the two former the infection is capable of experimental propagation; in the latter two the nature of the poison is as yet an inferential matter. All four present a preponderance of motor symptoms. Tetanus and hydrophobia are properly surgical conditions, and will be very briefly outlined.

TETANUS.

Tetanus is an acute infectious disease marked by tonic spasms of the voluntary muscles, usually commencing in those of the jaws; hence the names *trismus* and *lockjaw*.

Etiology.—The disease is comparatively more common in hot climates and in the colored races than among Caucasians in temperate and cold regions. This may have relation to the better protection by footwear and clothing in the latter conditions. It spares neither age nor sex, and is a common disease among horses. It may occur endemically. It is probably always introduced traumatically, and can usually be traced to inoculation by objects contaminated by the ground-soil, in which the bacillus of tetanus readily lives. Naturally, the hands and feet are the most common locations of such contaminated abrasions or more extensive lesions. The bacillus first discovered by Nicolaer, and cultivated by Kitasato, is an anaërobic, drumstick-shaped, motile microbe. Culture filtrates contain tetanizing poisons which are active when inoculated, but not when ingested. Experimental evidence indicates that, like strychnin, their action is mainly upon the spinal cord.[1]

Morbid Anatomy.—The condition of the wound presents nothing characteristic, and in the brain and spinal cord the congestion, perivascular exudation, small hemorrhages, and pigmentation of cells sometimes encountered are neither constant nor distinctive. They may even be looked upon as the results of the spasmodic conditions that mark the clinical course of the infection. The same is true of the serous ecchymoses, pulmonary congestions, and muscular ruptures.

Symptoms.—From two to twenty days or more after inoculation the first symptoms appear. The intensity of the disease and its fatality are usually in direct proportion to its early onset. Stiffness of the neck- and jaw-muscles first appears, limiting mastication, the movement of the tongue, and of the head. Malaise, chilly sensations, or rigors may antedate the muscular stiffness, but usually do not attract much attention.

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[1] Wasserman and Takaki, "Berlin. klin. Wochens.," June 3, 1898.

The muscular spasm increases in intensity and invades the face and trunk. From the tonic action of the zygomatic group the angles of the mouth are retracted in the characteristic sardonic grin that uncovers the teeth, and the jaws can be only slightly separated or may be quite immovable. The head is then retracted, and the entire back may be affected, causing rigidity, or, in greater degree, opisthotonos. In some cases the trunk is bent laterally or forward. The lower extremities are usually more affected than the upper, and the forearm and hands are last and least involved. The muscular spasm is chiefly tonic, but if the condition becomes well marked there are sharp, short, convulsive exacerbations that may reach a most frightful intensity. They are then provoked by the slightest irritation, such as a sudden noise, a bright light, a touch, the jarring of the bed, or any mòtor effort. They may occur a few times a day or, in extreme cases, with great rapidity, at scarcely noticeable intervals. When these develop they are attended by pain proportional to their intensity and duration. They may impede the thoracic respiratory excursions or induce laryngeal spasm and dyspnea or asphyxia. Often the thoracic or laryngeal spasm induces a hoarse noise, which, taken with the distorted face, rigid limbs, retracted head, and opisthotonic position, presents a frightful picture. Profuse perspiration may be occasioned. The temperature may be normal, slightly increased, or hyperpyrexia may appear and ordinarily precedes a fatal termination. Through it all the mind remains unclouded.

Varieties.—*Head tetanus or cephalic tetanus* follows wounds upon the head, face, or neck; is usually of prompt appearance after the inoculation; is ordinarily marked by trismus, dysphagia, facial palsy, and respiratory difficulty, a rapid course, and a fatal termination. The modification of tetanus in this form appears to be due to early poisoning of the medulla. The facial palsy that frequently and the oculomotor palsies that sometimes occur indicate nuclear disturbance. The difficulty in swallowing gives a rough resemblance to rabies and has led to the term *tetanus hydrophobicus*. *Tetanus neonatorum* is usually due to infection of the umbilical stump, and is unknown to aseptic midwifery. *Puerperal tetanus* occurs in parturients. The invasion route is usually through the uterus.

Diagnosis.—Given a locus of inoculation, the disease can scarcely be mistaken. When a history of trauma is wanting *hydrophobia* may be suspected, but lacks the jaw-spasm and persistent muscular rigidity. *Strychnin poisoning* is a closer imitator, but has a more rapid onset, more violent and extensive convulsions, trismus is absent, and relaxation occurs between the spasms. *Tetany* affects the hands and feet mainly and primarily and shows a number of special reactions, such as increased electrical excitability and Trousseau's sign. *Hysteria* may imitate tetanus, but ordinarily gives a hysterical history and presents the stigmata of the neurosis. It also usually appears suddenly after a hysterical convulsion, suddenly disappears and recurs, and lacks the nuchal rigidity and mental clearness of tetanus. Bacteriological examination of pus from wounds may make or confirm the diagnosis.

Prognosis is always grave and the mortality is over eighty per cent.

Cases appearing before the sixth day usually die; those appearing after the twelfth day are likely to recover. Death results from apnea and heart-strain. Favorable indications are: late onset, limited muscular spasm, absence of respiratory and medullary symptoms, infrequency of convulsions, normal temperature, and ability to receive and assimilate nourishment.

Treatment.—If the wound of entry for the tetanus infection is in an unhealthy state, surgical measures of local disinfection are always in order, and usually consist of scraping, cauterization, and the employment of active germicides. Lambert[1] believes that hydrochloric and carbolic acid together furnish the best local application. It often happens that the infection atrium is completely healed, and even early cauterization seems to be of doubtful assistance in checking the disease. The *general management* is of prime importance. The patient should be secluded in a darkened room and every possible excitation be prevented. Alimentation should be carefully maintained by easily digested fluid foods, and, if necessary, by the use of the nasal tube or by rectal injection. Sedative drugs and antispasmodics are indicated, and various ones have cures credited to them. Chloroform and nitrite of amyl are useful to meet the convulsions. Chloral, bromids, morphin, calabar bean, and curare are advised, but must be used with a free hand or omitted entirely. Hot baths sometimes act most soothingly. Active artificial respiration is required in case of dyspnea and threatened asphyxia. *Immunization* of late years has been attempted by the use of the antitoxins introduced by Tizzoni and Catani. They have been found practical and reliable in animal experiments. There is much diversity of opinion regarding their value in human tetanus. Kneass,[2] from a tabulation of sixty-one cases treated by tetanus antitoxin, finds an insignificant advantage over the older medicinal methods, and Berger, Roux, Yandell, and others are of the same opinion. Lambert[3] states a mortality of thirty-seven per cent. under the antitoxin in acute cases developing within eight days of the infection. It is not unlikely that the antitoxins will be so much improved as to give better and more reliable results, and they can not well be omitted in the treatment of this disease. They are valuable in proportion to their early use. Antitoxin has been injected into the substance of the brain through trephine openings in order to bring it quickly into operation, but the results hardly warrant the method. Intraspinal, subdural, intravenous, and subcutaneous injection are less objectionable and probably equally efficacious. The removal of spinal fluid and the intradural injection of eucain and morphin has been of apparent benefit. At present, medicinal preparations are also imperatively demanded in the treatment of tetanus.

HYDROPHOBIA.

Hydrophobia is an acute, infectious disease of carnivorous animals, transmissible to man and to other animals by inoculation. It is also known as *rabies* and *lyssa*. The inoculating animals usually are dogs or wolves, but the cat, skunk, and even poultry may carry the disease. The exact nature of the poison is unknown. It undoubtedly is a living contagium. The disease is rare in this country, but seems to be growing more common in the Eastern States, and almost invariably, in man, is the result of bites by rabid dogs. In North Germany, where the muzzling of dogs is rigidly enforced, the disease is almost unknown. It is, therefore, in civilized countries a preventable disease.

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[1] "Amer. Jour. Med. Sciences," Aug., 1897.

[2] "Jour. Am. Med. Association," July 18, 1896.

[3] *Loc. cit.*

Morbid Anatomy.—The nervous system frequently shows lesions, but these may be completely lacking and to some extent, when present, are secondary to the disease, following the spasms, dyspnea, and cardiac failure. They consist essentially of vascular disturbances: dilatation, perivascular leukocytal infiltration, ante-mortem intravascular clots, and minute hemorrhages. Such changes are most frequently encountered in the cortex cerebri, the medulla, and cord. According to Gowers, they are most intense in the neighborhood of the pneumogastric and hypoglossal nuclei. The perivascular infiltration in this locality may be intense enough to suggest miliary abscesses. The salivary glands and kidneys frequently show a similar infiltration and the mucous membrane of the pharynx and larynx is commonly congested.

Symptoms.—*Incubation* requires a variable period of from two weeks to six months, and there are reported cases occurring twelve and even eighteen months after inoculation, the virus having remained dormant. The ordinary incubation period is six weeks to two months. The length of incubation time, according to Horsley, is modified by a number of factors: (1) It is shorter in children than in adults. (2) Wounds of the face, neck, head, and hands, and the unclothed parts are especially dangerous, and the disease then develops promptly. (3) Punctures are the most dangerous; lacerations are serious in proportion to their extent. (4) The bites of rabid animals are serious in this order: Wolf, cat, dog, and other animals. About fifteen per cent. of the persons bitten by dogs known to be rabid develop hydrophobia.

Rabies varies in intensity in both animals and man. In cases of great severity paralytic features develop early, there is little excitement, and death promptly supervenes. When the poisoning is less profound, the disease runs a longer course, and presents a period of great motor and cerebral excitement. In man the *invasion* of the disease is frequently marked by irritation about the wound, with pain or numbness. Usually there is headache, depression, loss of appetite, irritability, sleeplessness, and anxiety. The pulse and temperature may be slightly increased. Bright lights, noises, and slight excitement of any sort are shunned owing to the increased sensibility. Stiffness of the throat-muscles and difficulty in swallowing are noticed. *A period of excitement* then usually develops, when, in rare instances, the central apparatus may be overwhelmed and the paralytic form, with ascending paraplegia and heart-failure, terminates the case within a comparatively few hours. In the excited period there is great motor restlessness and hypersensibility: spasms affecting the throat are induced by any fictitious stimulus, and swallowing becomes impossible, so that fluids are shunned, and the sight of them may even become unbearable; hence the name, hydrophobia. Noises, lights, a breath of air, may provoke the spasm; and it may involve the larynx and thorax, producing dyspnea, cyanosis, and an alarming asphyxia that even tracheotomy may fail to relieve. The respiratory and deglutition spasms are often attended by hoarse sounds and peculiar noises, that have been thought by excited laymen to resemble the barking of dogs. Saliva accumulates in the mouth or drules from its corners, partly from an increased secretion, but mainly from difficulty in swallowing. Hallucinations and delirium are frequently present, but the mind may be quite clear and the patient quiet between the paroxysms. The temperature commonly ascends to 100° or 103°, but the disease may be afebrile throughout. The spasms gradually become wide-spread and tetanoid, but rarely affecting the jaw and face, and relaxation occurs in the intervals. After one to three days they are followed by the paralytic period, and spasms no longer occur. An ascending paraplegia is commonly presented. Quiet, stupor, and coma follow; the heart's action becomes greatly weakened, and death follows syncope.

Diagnosis.—The diagnosis of a well-developed case, with motor excitement and a history of a bite by a rabid animal, can be readily made. We have to exclude *tetanus*, in which there is usually a very recent trauma, masticatory spasm, constant rigidity, and little or no difficulty in swallowing. *Lyssophobia* is a hysterical condition occurring in a neurotic, and is a variety of hypochondriasis with the fixed idea of rabies. The attacks that occur are hysterical exaggerations of the newspaper accounts of rabid patients, and such cases can usually be deciphered by the concomitant indications of hysteria. Every such case should, however, be carefully watched if it is reasonably certain that the patient has been bitten by an animal suspected of rabies. In every case it is also important to diagnose the condition in the alleged rabid animal, and this can be done with certainty if inoculation experiments can be made. In case of a bite by a dog, the animal should be placed under observation, if possible, and not destroyed. The saliva being highly poisonous, a little of it may be inoculated into the dura of a dog or rabbit, and precise results obtained. In dogs, the paralytic form of rabies is the most common, the furious form exceptional. The early symptoms in the dog consist of dullness, irritability, and surliness, the bark becomes metallic, food is neglected, but bits of wood, dirt, and other indigestible articles are sometimes swallowed. If furor develop the dog runs, snapping at everything in its way, especially attacking other dogs. Children, being unaware of danger and less able to escape, are more frequently bitten than adults. After a day or two, or a few hours only in some cases, paralytic symptoms set in. The animal becomes uncertain on its legs, the hinder limbs give way, and the paralysis becomes general, ending in death. The excited stage is commonly transient, and paralytic conditions promptly develop.

Treatment.—An inoculated wound, or one reasonably suspected, should be treated locally, at the first possible moment, by thorough cauterization, or excision, if practicable. The use of a ligature above the wound when on an extremity, suction, and free bleeding are also of value. The actual cautery, a bunch of burning matches, strong carbolic or nitric acid, may be used. *Immunization* by Pasteur's method is now practicable in most large cities. The sooner it is undertaken, the more successful it is likely to be. Less than one-half of one per cent. of all cases treated in this way have died of rabies. When hydrophobia develops in man, about the same treatment is required as in tetanus. There is no danger to attendants, and no case is on record where the disease has been transmitted from man to man. Forcible physical control is practically never needed. If food and drink can be swallowed, it should be freely given, but rectal alimentation is usually required. Chloroform, bromids, morphin, and sedatives are palliative only. The disease is invariably fatal.

TETANY.

Tetany, or *tetanilla*, is marked by peculiar tonic, bilateral, paroxysmal, muscular contractions, commencing in and usually confined to the extremities, and presents an increased mechanical and electrical excitability of the nerves. It may occur in epidemics, is endemic in many places, and commonly develops on a background of malnutrition.

Etiology.—It occurs most frequently before twenty and practically never after fifty years of *age*. Both *sexes* are affected, males more frequently before adult life, females more commonly thereafter. In Paris, Berlin, Prague, Vienna, and in Syria it may be considered *endemic*, and *epidemics* of it have been noted in these places, and in schools and garrisons. In the neurological clinics of Berlin [1] tetany furnishes one per cent. of all cases. In Vienna seven-tenths of one per cent. It appears most commonly from *January to May*. In America it is comparatively rare outside of quarters in large cities largely populated by foreigners. *Predisposing causes* include nearly every variety of depraved nutrition. Sarbo,[2] from an extensive review of the causes of tetany, asserts that impaired nutrition is the only common factor. Of most importance are chronic gastro-intestinal disorders, especially gastric dilatation, with hyperacidity, fermentative diarrhea, and chronic constipation. Rickets is a very common accompaniment in children. Cassel [3] found it in 58 out of 60 cases. Tetany may follow acute infectious and septic processes, or appear during pregnancy or lactation. Enucleation of the thyroid has been followed by tetany in a considerable proportion of cases. Billroth and Wölfler reported 19 cases of tetany in a total of 123 thyroidectomies. There is a certain relation between tetany and myxedema, which may concur in a given case. If about one-fifth of the thyroid is spared, tetany does not appear, according to von Eiselberg. Murray insists that it does not occur if the parathyroid is spared. As *exciting causes* of the paroxysmal attacks, exposure to cold and emotional disturbances are often active, as are overexertion, exhaustion, acute diarrhea, and vomiting. The administration of ergot, chloroform, and alcohol has caused them to appear, and they may be induced by mechanical irritation of the nerve-trunks and blood-vessels, in the manner to be described later.

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[1] Lathrop, "Boston Med. and Surg. Jour.," Nov. 19, 1896.

[2] "Deut. Zeit. f. Nervenheilk.," 1896.

[3] "Deut. med. Wochen.," Jan. 28, 1897.

The epidemic, endemic, seasonal, remissive, and toxic features of the disease point very strongly to the activity of some zymotic agent, but as yet it has escaped detection. Oddo[1] suggests that it may only be active in the presence of some special form of perverted digestion. The vulnerable portion of the nervous system appears to be the spinal and bulbar gray and the peripheral nerves. *Morbid anatomical* changes are not constant, though suggestive. Cloudiness and swellings in the anterior horns have been noted by Weiss and by Barome and Cervasato. Peters,[2] in a recent communication, claims to have found in seven cases an acute inflammation of the extradural connective tissue among the extradural bloodvessels and fat, constituting a pachymeningitis externa and secondarily inducing a root neuritis and ganglionitis proportionate to the clinical manifestations of the disease.

Symptoms.—The clinical manifestations mainly fall within the domain of the lower motor neurons. The first thing to attract attention is the development of spasmodic stiffness, usually first appearing in the fingers and wrists. After the recurrence of several attacks adults sometimes recognize in malaise, headache, depression, and general pains, premonitions of the on-coming rigidities. The attacks are initiated by a feeling of prickling, numbness, and some local pain. The spasm comes on slowly and increases gradually in intensity, accompanied by growing discomfort and pain in the muscles. There are no mental features attributable to tetany. The spasms begin peripherally in the fingers and toes, and advance toward the trunk. Ordinarily, they are limited to the limbs, and mainly affect the parts below the elbows and knees. The upper extremities may alone be invaded. In other cases the tonic spasm reaches the roots of the limbs and invades the trunk, and may involve all the body-muscles in very severe cases. Retraction of the head and strabismus are seldom encountered. The contraction is tonic in character and usually persistent during the attack, but may intermit. The attack may last from a few minutes to many hours and occur several or many times daily. Attacks may cease for intervals of days, weeks, or months, and then reappear, but if latent they can be provoked in the intervals by appropriate manipulation.

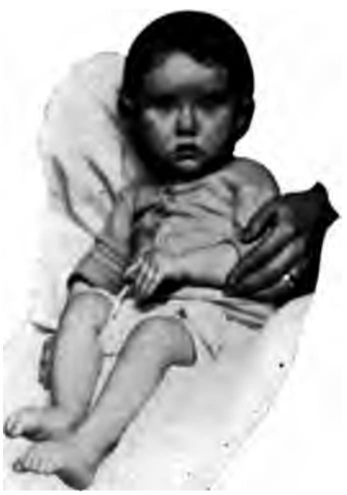

Fig. 233.—Infant with mild attack of tetany, showing characteristic spasmodic position of hands and feet.

The positions and attitudes caused by the spasms of tetany are strikingly peculiar. Ordinarily, the *hands* are rolled into the cone-shape of the accoucheur, the digits flexed at the metacarpal joints and rigidly extended at the internodal articulations, the thenar and hypothenar eminences approximated. The *wrist* may also be flexed and the hand drawn to the ulnar side. Less commonly the fingers and wrists are extended, or the hand may be made into a fist grasping the thumb, or the thumb may protrude between the index or middle digits. In cases of severity the *elbows* are flexed and adducted strongly against the body. The *feet*, when affected, present a forced equinovarus position, with flexed and sometimes overlapping toes. In extreme cases there is flexion at the *knees* and *hips*. The *muscles* of the forearms, legs, hands, and feet are tense, firm, and often sensitive. Voluntary movements in the parts are impossible, and passive motion causes pain. When the spasm invades the trunk, intercostal, abdominal, and spinal rigidity may appear and *breathing* be impeded. In rickety children *laryngismus stridulus* is not uncommon; in adults laryngeal spasm constitutes a serious complication. In rare instances spasm in the neck-muscles draws down the chin and the angles of the mouth, and has even produced fatal compression of the air-passages. Solovieff[1] has noticed in adults rhythmic contractions of the *diaphragm* synchronous with the heart-beat and attended by a whistling sound in the left lung. Thoracic and diaphragmatic rigidity may induce *asphyxia* and even death. Ordinarily, the face escapes. The *sphincters* may be tonically contracted, inducing obstipation and anasarca, according to Oddo and Sarles,[2] who also noted indican in the urine, due to intestinal fermentation, and confirmed Weiss' observations of the hypertoxicity of the urine. Retention of urine may be the most prominent symptom.[3]

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[1] "Münch. méd. Wochen.," Nov. 10, 1896.

[2] "Deutsch. Arch. für klin. Med.," Vol. lxxvii.

The electrical and mechanical irritability of the motor nerves is peculiarly increased. If pressure be made over the median or ulnar nerves, the spasm in the hand is increased, or during its absence is provoked. Pressure over the brachial artery may have the same effect. This is known as *Trousseau's sign*, and is practically pathognomonic. Gentle tapping on the nerves, as with a percussion hammer, has the same effect. Chvostek discovered that the facial nerve could be aroused in the same manner, causing a facial contortion exactly limited to the distribution of a branch or of the entire nerve, depending upon the location of the blows—*Chvostek's sign*. Erb first described a peculiar exaltation of the electical excitability, especially to the galvanic current—*Erb's phenomenon*. A single cell, giving but one or two milliamperes of current, may be sufficient to provoke sharp contractions, and anodal opening tetanus (A. O. Te.) is found in this disease alone. Sarbo has, in a single case, noted the myotonic reaction in the triceps, a response that was considered confined to Thomsen's disease. It consists of persistent contraction, lasting some moments after cessation of the galvanic current. The faradic responses are also accentuated in most cases, but may remain about normal. *Pressure* upon the nerves is more than ordinarily painful, and readily induces persistent paresthesia in their cutaneous distribution. The tapping that causes spasm also induces pain. *Edema* of the hands and feet and localized perspiration may be encountered. The *temperature* may be normal, but is often elevated, as might be expected, in the gastro-intestinal cases, and subnormal in the arthyroidal state of operative myxedema. Auditory and optic symptoms and trophic changes in the muscles are found only as accidents. Objectively, *cutaneous sensibility* is normal and the *reflexes* are unchanged except when inhibited by the spasmodic state of the muscles.

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[1] "Rousski Vratch," 1902.

[2] "La Méd. Inf.," Sept. 15, 1894.

[3] Burkhardt, "Jahrsb. f. Kinderh.," 1899.

Course.—The great majority of cases run a mild course to recovery in a few weeks, especially if the underlying cause can be removed, but when tetany develops on gastric dilatation or chronic catarrhal enteritis it is likely to have a protracted course. Appearing generally when the organism is already depressed by malnutrition, it may be a formidable complication, and in the severe cases, where the spasms invade the trunk and implicate the respiratory apparatus, death by asphyxia may result. In pregnancy it is likely to be mild and usually terminates promptly after delivery, but parturition may cause very severe exacerbations of the attack. The lactational cases are usually manageable if the child be weaned and the nutrition of the patient reëstablished. Tetany in the athyroidal state is frequently fatal; it may be permanent and exceptionally it yields to thyroid feeding. Only when Trousseau's and Chvostek's signs fail can the disease be considered at an end. In fatal cases the spasms increase in frequency, distribution, and intensity, and asphyxia destroys the patient, but death in tetany is more commonly the result of the underlying disease.

Diagnosis.—The diagnosis is easy if the characteristic spasms are accompanied by the mechanical and electrical overexcitability of motor and sensory nerves. We have to exclude *tetanus*, in which the spasms are preceded by stiffness in the masseters which does not subside in the intervals between the spasms. It is also marked by nuchal rigidity and great general irritability. In tetanus the spasm is propagated toward the extremities, usually sparing the hands; in tetany it is centripetal and usually begins in the hands. The phenomena of Trousseau, Chvostek, and Erb are absent in tetanus. *Hysteria* also lacks these special signs and has its own stigmata. *Meningitis* may be mistaken in infants, but presents none of the special signs of tetany.

Prognosis.—The prognosis is usually good, but hangs upon the nature and manageability and the underlying cause. It is unfavorable in proportion to the extent and degree of malnutrition present. It is grave in the athyroidal cases, which present a mortality of about eighty per cent. A tendency to recurrence upon renewal of the predisposing state is marked. Thus, some women present tetany in several successive pregnancies, and exacerbations of the predisposing gastro-intestinal state may incite a return of the tetany. Extreme severity of spasms, involvement of the respiratory apparatus, intense manifestation during labor, and cerebral symptoms, as in uremic cases, are of serious import.

Treatment must be directed to the removal of the underlying *causal state*, but it is rarely necessary to interrupt pregnancy. Rickets, gastric dilatation, intestinal catarrh, intestinal parasites, and lactation have their several indications. Hygienic and sanitary conditions must be attentively studied. The *spasms* are controlled by quiet, rest, and warm baths. Bromids and chloral are useful in the interval, but morphin, and even chloroform, may be required for the attack. When this coincides with labor, the birth must be hastened. Chloroform must be used with circumspection, as it may provoke laryngeal spasm, as may also the employment of the stomach-tube in gastric cases. In the athyroidal cases thyroid feeding sometimes gives relief, but can not be relied upon. The administration of thyroids in other cases may also be tried. Massage, electricity, and passive movements must be avoided, as they usually intensify the spasms when present, and provoke them if absent. A hot sponge over the larynx may relieve laryngeal spasm, but tracheotomy may be required in rare instances. Oddo [1] attaches much value to the continuous use of calomel, which corrects the gastro-intestinal fermentation and expels toxic substances. Dietetic regulations are often of the first importance. The condition of the bladder should be watched and excessive retention of urine prevented.

CHOREA.

It is desired to limit the term chorea to a definite morbid entity. The choreiform features of hysteria which mark epidemics of St. Vitus' dance, hereditary chorea, or the so-called chorea major of Huntingdon, the electric chorea of Dubini, habit chorea or the maladie des tics, and the various forms of myoclonus should be carefully distinguished from *minor chorea* or the *chorea of Sydenham*, with which we are now to deal. It is commonly called St. Vitus' dance, but that term may better be reserved for the hysterical forms.

"Chorea," judicially says Osler,[2] "is an acute disease of childhood, rarely of adults and of the aged. Characterized by irregular, involuntary movements, a variable amount of physical disturbance, and associated very often with arthritis and endocarditis. The disease is usually considered as a neurosis, but the clinical characters of the severe cases and the frequent heart and joint complications have suggested to many recent writers that it may be due to a specific poison."

Etiology.—*Predisposing Causes*—*Sex.*—Girls are affected somewhat more than twice as frequently as boys, and the disproportion becomes even greater after puberty. The vast majority of cases develop between five and sixteen years of *age*. Chorea is comparatively rare before the age of four. The reported congenital cases are usually mistaken instances of organic cerebral disease. After twenty the disease is most rare, but may appear in advanced years. The great majority of reported senile cases, however, are instances of motor disturbance symptomatic of organic brain lesions. It is somewhat more common in unhygienic and cramped conditions of life, and hence in urban communities, but spares no social grade or locality. Chorea is extremely rare in the dark-skinned *races* on this continent. Negroes and Indians of full blood are very seldom affected. The *seasonal relations* of the disease are most interesting. According to Lewis, the frequency of chorea reaches its lowest curve in November, but rises rapidly in December, remains stationary in January and February, mounts still higher—to its acme—in March, falls in April, again rises in May, and then gradually declines to its November starting-point. This trace corresponds with that of rheumatism, the general amount of sickness, and climatic vicissitudes. The *neurotic make-up* plays a distinct predisposing rôle, so that we learn to expect a history of various neuroses and psychoses in the family, and of "nervousness" in the patient. Choreics commonly show some of the so-called stigmata of degeneracy and often give a history of pavor nocturnus, enuresis, infantile convulsions, febrile deliriums, impressionability, and mental precocity. It is not rare to find that the mother has had chorea.

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[1] *Loc. cit.*

[2] On "Chorea," Philadelphia, 1894.

Inciting Causes.—Fright, worry, especially at school; *mental shock and strain* generally, and *overstudy* particularly, are frequently alleged inciting causes, but on close analysis usually retain but little significance. Very frequently the early mental and even motor symptoms of the disease and of rheumatism will be found to have antedated the psychic trauma that precipitated the choreic storm. *Imitation* plays a most insignificant, if not entirely negative, rôle. A case of true chorea in a school or home for children may, however, start an epidemic of hysterical St. Vitus' dance, or of hysterical rhythmic spasms. Hysteria may, indeed, ape chorea to the minutest detail, and they may be associated. *Traumatism* can not be dissociated from mental shock, and parents always assiduously seek for some such incident and cause. Its real value as an etiological factor is difficult to estimate, but seems slight, and often the particular injury is but one of the insignificant mishaps of every-day life in childhood. *Reflex irritation,* prominently urged by the older writers, arising from the intestines, and particularly from worms, is seldom found, but should not be overlooked. Diseased conditions in the nasopharynx are more likely to induce tics than chorea, and the same may be said of eye-strain, the fetich of some recent writers. If the ocular apparatus is unbalanced or refractive errors are found, they should be relieved here as well as elsewhere. It is not unreasonable to suppose that their deleterious influence may protract the nervous manifestations of chorea, or may predispose to it by lowering the general tone. *Pregnancy* appears capable of causing a recurrence of chorea or of favoring its development and modifying its course. The chorea of pregnancy furnishes one of the varieties of the disease, and accounts in part for the larger proportion of female cases in adult years. As strongly urged by Tourette, many such cases are purely hysterical, though commonly considered choreic. Chorea is sometimes preceded or accompanied by the *infectious diseases* of childhood, as might naturally be expected, given the fact that chorea is especially a malady of early life. The relationship is mainly one of coincidence. They may, however, modify the chorea. Occurring during its early period, they tend to intensify it, but if it be on the wane, they seem not rarely to hasten its regression. The favorable influence, if it exists, may perhaps be attributed to the forced rest they necessitate, and we are to see that rest is the most important element in the treatment of chorea. *Anemia* and *malnutrition* sometimes precede chorea, and may furnish a certain liability to its invasion, but usually they follow its development and are symptomatic of it.

Rheumatism (*Cardiopathy, Arthritis*).—The questions arising regarding the relations existing between rheumatism and chorea are interesting and practically important. In order to start aright, it should be stated that rheumatism is here meant to broadly signify the acute manifestations of an infectious process that is marked by inflammatory disturbance of articular, serous, and endocardial surfaces. This is presumably a heteropathic disorder, but in most instances due to the activity of an unknown specific micro-organism; unless we accept as such the streptococcus isolated by Triboulet, Wasserman, Pain and Poynton, and Benton and Walker. It has long been observed that acute rheumatism and cardiac lesions may precede, attend, or follow chorea. Different observers find such relation in widely varying ratio. One sees rheumatism in "growing pains"; another requires that all the articular manifestations should be present to justify its recognition. The most reliable statistics give a rheumatic association in about 20 per cent. of all cases. Thus, Osler, 21 per cent. and 18.24 per cent. in two series; Townsend, 21; Crandall, 54; Starr, 18; the British Collective Investigation, 22 per cent. Usually the arthritis precedes the chorea, rarely it follows, and exceptionally it develops during the progress of the motor disturbance. If the milder manifestations of rheumatism are accepted in this connection and allowances made for the unrecognized cases, it is permissible to say that it is associated with chorea in one-half of all cases. Fatal cases of chorea almost invariably show endocardial vegetations which differ in no particular from those due to rheumatic endocarditis, yet such cases give a rheumatic history in only about twenty-five per cent.

Pathogenesis.—It is needless to even enumerate all the theories that have been advanced regarding the nature of chorea. Those now advocated may be divided into the neurotic, the infectious, and the rheumatic theories. Granting that acute rheumatism is an infectious disease, the third division is embraced in the second. The neurotic theory is based largely upon the appearance of the disease during the years of active growth, the common neurotic peculiarities and antecedents of the patients, the incitement by mental shocks, the psychical symptoms, and the complexity of the nervous disorders, which embrace motor, reflex, and sensory troubles. Even the arthritis has been referred to a nervous source. Charcot said it was the old question of arthritism in combination with nervous diseases. Joffroy denominates chorea as a cerebrospinal neurosis of the period of growth.

The theory of infection rests upon the influence of age, sex, and season; the association of chorea with other infections, its intimate relation with rheumatism; the infectious aspects of fatal cases clinically, and the presence of endocarditis at practically every autopsy, often accompanied by pericarditis, pleurisy, parotitis, abscess formation, and other septic processes. Finally, there are those who, following Kirkes, Roger, and others, see in chorea only a manifestation of rheumatism. This theory of identity is recently maintained by Churton.[1] He says: "The postulated toxin (x) being accepted as an essential element in the causation of rheumatism, depressing conditions (y) determine the first position or locus (z) of the disorder. . . . If (y) is a fright, shock, or intense excitement, (z) will be the nervous system; in the developing brain of a child the result is usually chorea; in adults it may be delirium or coma, perhaps hyperpyrexia. . . . Wetting of the feet always causes arthritis first in the lower extremities; of shoulders, in the upper extremities." The embolic theory of Kirkes, upheld by the experiments of Money, was based on the supposition that endocarditis always preceded chorea, which can not be maintained.

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[1] "British Med. Jour.," Sept. 19, 1896.

As to the precise *location* of the disease in the nervous system, the mental symptoms, frequent monoplegic or unilateral distribution of the chorea, and the practical exemption of the trophic functions of the lower motor neuron, with absence of sensory disturbance, point to the cerebral cortex. Wood, from experiments and investigations in canine chorea, located the disturbance in the spinal gray, but this disease is not identical with human chorea.

Regarding the nature of the *specific microbe* in chorea, nothing definite can now be offered. In 1891 Pianese [1] cultivated a rod-shaped microbe from the cord and brain of a fatal case of chorea, which, injected into animals, produced apathy, then tremor, then convulsive movements, and, finally, death. Autopsical search in these animals detected this bacillus exclusively in the nervous apparatus. This finding has not been confirmed. Dana [2] has found a diplococcus in the meninges of a fatal case, but as subacute leptomeningitis was present, it was possibly or probably the specific micrococcus of that disease. Various streptococci have also been noted. Westphal, Wasserman, and Malkoff [3] isolated from the blood, brain, and cardiac vegetation of a fatal case of chorea a micrococcus which produced arthritis in animals.

Morbid Anatomy.—As chorea is seldom fatal, the post-mortem changes found in the extraordinarily severe cases that result in death can hardly be supposed to fairly represent the anatomy of the disease. Even in such cases there are no uniform or characteristic changes. In the *brain and spinal cord* intense hyperemia, peri-arterial exudations, minute hemorrhages, softened spots, and occasional emboli have been noted, especially in the deeper portions of the motor tracts in and about the basal ganglia. The *chorea corpuscles*, first described by Elischer, and often found by others, are equally developed in various infections, as proven by Manasse, who found them in twenty autopsies upon septic subjects, and was able to produce them in dogs by putrescent intravenous injections. They are hyaline in chemical reaction, develop in and around the arterioles and in the perivascular spaces throughout the brain and cord, and furnish another argument for the infectious character of chorea. Turner [4] describes swellings and opacities in the cortical pyramidal cells. One of his five cases had puerperal sepsis and two albuminuria, both of which might account for the cellular changes. The two remaining cases, if identical, may be taken as showing changes of a septic or infectious nature. The *heart* is more often diseased in the fatal cases of chorea than in any other malady whatsoever. Osler, in the 73 cases collected by him, finds cardiac lesions recorded in over 90

[1] "La Riforma Med.," July 14, 1891.

[2] "Amer. Jour. Med. Sci.," Jan., 1894.

[3] "Berlin. klin. Woch.," No. 29, 1899.

[4] "Path. Soc. Trans.," 1892, vol. xliii.

per cent., consisting of recent endocarditis, 62 cases, with pericarditis 19 times; pericarditis alone in 2 cases; chronic mitral endocarditis twice, and fatty heart once. The ordinary endocardial appearance is a row of papular granulations at the mitral orifice. All varieties of incidental and accidental septic conditions are reported in the literature. Staphylococcus aureus and pyogenes have been noted by Guizetti, staphylococcus aureus and streptococcus by Reichardt. Preoprajensky[1] claims to have found streptococci in the blood in two cases, both of which were benefited by the use of antistreptococcus serum. Benton and Walker[2] claim to have found a specific streptococcus in chorea and acute rheumatism reacting in certain respects differently from the streptococcus of human septicemia.

Symptoms.—Chorea is commonly of insidious *onset*, but its motor symptoms may appear abruptly—that is, within a few hours or days after a fright or other mental disturbance. In the large majority of cases parents can easily recall that for a period of days or weeks before motor disturbance attracted their attention the child had been *peevish*, obstinate, apprehensive, easily displeased, and less companionable with its playmates. Perverseness and moral obliquities sometimes appear. Very commonly the *sleep* has been disturbed and broken by distressing dreams or actual nocturnal pavor. More often there is mere restlessness and difficulty in getting to sleep. This *prodromic period* should be carefully investigated, as it is likely to give valuable warning in case of recurrent attacks. At school the child becomes inattentive and forgetful, and finds increasing difficulty with its studies. If reprimanded, there is undue depression or unusual emotion or capriciousness. The inability to study, due to the lack of mental concentration, leads too often to the supposition of overstudy; but it is particularly among the bright, precocious, and easily stimulated school-children that chorea is likely to appear. These are exactly the children whose mental development should be retarded, and it is among these that forcing methods in school-work are most baneful.

After a widely varying period the *motor choreic features* appear. These at first consist ordinarily of sudden, unwilled, slight movements or unexpected relaxation of muscular contraction. The child appears maladroit, upsets its cup, drops articles from its hands, and lays itself liable to admonition, and, unfortunately, in some instances, to chastisement, which may bring down the choreic storm in a burst of motor twitchings, grimacings, and spasmodic disturbance. Lacking cause of sudden exacerbation, the movements gradually become more pronounced and bizarre. There is often complaint of *fatigue*, *pains* in the limbs or joints, and frequently *loss of appetite* and *constipation* are early noted.

Motor Features.—The choreic movements ordinarily begin in the muscles of the hands and forearm, but the face is soon, sometimes first, affected, and then the lower extremities, shoulders, and trunk, in varying order. Occasionally, the chorea is confined to a single extremity or to one side of the body, or, beginning on one side, may invade the other, subsiding or persisting in the original territory. Commonly, both sides are not equally involved. As a rule, the affected muscles show three important functional modifications: (1) Unwilled but conscious twitchings or spasm; (2) inability to maintain steady contraction, and (3) loss of power. The choreic movements vary not only in distribution, but in intensity at different times and in different cases. We may first consider a case of average severity. The spontaneous actions may be described as disordered, irregular, arrhythmic, of considerable amplitude, and of a rapidity between tics and athetosis. They cease during sleep, though there may be great restlessness. They may even prevent sleep. They can be slightly controlled during the execution of voluntary movements, but are excited and exaggerated by embarrassment and any emotional excitement. Mitchell and Rhein [1] define several varieties of choreic cases depending upon motor peculiarities: "(1) Cases which show, at some stage or throughout the attack, an absence of movements during rest. (2) Cases with continuous movements during rest, but increased by intentional effort. (3) Cases with severe choreic movements, entirely disappearing during muscular acts. (4) Cases in which the movements are unaltered by muscular efforts. (5) Cases presenting several of these phases at different times." In the *face* they usually cause bilateral grimacing, especially affecting the lips and nose, less frequently invading the muscles of the brow and eyelids. The lips may be quickly pursed up or retracted, the tongue protruded and retracted, the teeth snapped together. In this way speech is impaired and becomes halting or explosive, due entirely to faults of articulation, as the larynx is practically never involved. *Swallowing* is sometimes difficult, mainly, however, on account of the choreic movements of the lips, cheeks, tongue, and palate. The *tongue* is usually affected very early, and in perhaps the majority of cases the choreic movements persist in this organ after they have elsewhere disappeared. Ordinarily, if the patient is asked to show his tongue the chorea is at once provoked in the facial muscles, but the tongue, too, is animated by involuntary writhings that appear, subside, reappear, and usually end in its sudden retraction and the quick closing of the mouth. In very exceptional cases the *ocular muscles* are implicated, causing momentary diplopia, and movements in the iris have been seen attending momentary confusion of vision. Sluggishness of pupillary contraction to light is not uncommon, and a few cases of retinal embolism are recorded.[2]

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[1] "La Sem. Méd.," Dec. 10, 1902.

[2] "Br. Med. Jour.," Jan. 31, 1903.

In the *upper extremities* the choreic twitching is most and first developed in the fingers, which move individually or together, separate and close, extend or flex, with more or less disorder. Pronation and supination are more common than wrist movements, and the shoulders are more affected than the elbows. Indeed, it is rare that shrugging of one or both shoulders does not take place. When the chorea is well marked, objects are grasped with difficulty. The hand approaches them by zigzags and suddenly swoops down upon them. Finally, prehension may be impossible, and the patients can no longer feed themselves, or spill everything they attempt to carry to the mouth. The *lower extremities* are usually less affected, but, as in the upper members, the digits show the most disturbance, flexing, extending, or separating in disorder. The big toe and the thumb are most active, as a rule. The *station* is rendered unsteady, sometimes uncertain, and rarely impossible, by the movements of the legs and feet. In walking the *gait* is often peculiarly disturbed. The steps are unequal in length and irregular in rhythm. The feet may be jerked from the direct line of advance, raised too high, or brought down too vigorously, but never rhythmically. The knees are not always firmly supported, so that altogether there is sometimes presented a peculiar resemblance to the many-jointed action of marionettes danced on a string, which, taken with the grimaces and with the contortions of the hands, recalls the clownishness Sydenham so graphically described. The muscles of the *trunk* and *neck* do not escape, and may cause noddings and bendings that are often most apparent when the patient is seated. The *diaphragm* and the *thoracic muscles* are commonly invaded, causing irregularity of *respiration* and sometimes *spasmodic noises*, or there may be peculiar involuntary clucking or swallowing sounds. The choreic movements may be so continuous and severe as to prevent sleep, to confine the patient to bed, and to cause innumerable bruises and excoriations by friction or by rough contact with hard substances, such as the walls and furniture.

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[1] "Phila. Med. Jour.," Jan. 22, 1898.

[2] H. Thomas, "Johns Hopkins Hosp. Bull.," Oct., 1901.

If the patient is directed to grasp the physician's hand and hold firmly, *inequalities in pressure* will at once be observed. Relaxation or sudden increase of muscular tension, or both, are noticed. Of still greater importance is a *loss of muscular power*, which is practically always present in muscles affected by chorea, as can be clearly shown in the unilateral cases. This may reach a loss of fifty or seventy per cent., and in the paralytic form of chorea it constitutes the major motor difficulty. The paretic feature of chorea explains the ready fatigue and accentuates the need of rest in the management of the disorder. Russel[1] notes that the *handwriting* may be (1) merely choreic, (2) may be very good even when choreic disturbance is well marked, (3) may be almost impossible though choreic movements are extremely slight, (4) may be entirely unintelligible though control of the pen is good, and (5) may present pure motor agraphia.

The *sphincters* are never affected except in the last stages of the fatal cases. Objectively, *sensibility* is normal, as a rule, and any considerable anesthesia or dysesthesia should cause a suspicion of hysteria. Triboulet laid much importance on sensitive spots beside the spinal processes of the vertebræ and over the intercostal nerves, but they are inconstant. The *electric excitability* of the nerves and muscles is sometimes increased, and the anodal closing contraction may equal the similar cathodal response. General *loss of flesh* is common, but localized muscular *atrophy* is very seldom found. Shaw[2] calls attention to a jerky response upon eliciting the knee phenomenon, otherwise the *reflexes* are unaffected.

Mental Disturbances.—Aside from the temperamental, moral, and affective changes so common in the prodromal stages, and which may persist throughout the attack, other mental disturbances may rarely occur.

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[1] "Lancet," April 1, 1899.

[2] "Albany Med. Ann.," May, 1897.

The former are, in a sense, proper to chorea, and with the myasthenia may cause a marked change in the *facies*, which presents an inane and stupid appearance, often at great variance with the normal attributes of the child. Some of this may be due to the weakness of the facial muscles, but mainly it is consistent with the hebetude, irritability, and weakened mentality in such cases. *Hallucinations* of sight are frequently, and of the other senses rarely, noted. They usually appear toward or during the night. In the grave cases, when the *choreic status* is produced, the temperature elevated, and the muscular activity at its height, *delirium*, commonly of a hallucinatory character, is often present. Occasionally, *psychoses* of various forms are encountered—the concomitant manifestations of degeneracy.

Cardiac Disorders.—In chorea the heart frequently presents clinical symptoms, and apparently is much oftener involved than auscultation indicates. This is shown by the considerable percentage of cardiac lesions in fatal cases without cardiac symptoms, and the astonishing number of cases of chorea which show organic heart-lesions upon examination years after the chorea has disappeared. During chorea heart-murmurs are present in about one-third of the cases, and are functional or organic. In addition, there are disturbances of rhythm, rapid action, and pain. Galdi[1] calls particular attention to the variability of the cardiac diameters during chorea and their wide and rapid changes under insignificant causes.

Functional murmurs are most commonly heard at the base and to the left of the sternum, with the systole usually most intense over the pulmonary valves, and often attended by rapid heart-action. Anemic murmurs over the tricuspids propagated into the neck are not infrequent, and are often associated with an increased area of cardiac dullness. They may only be noticeable with the patient in the horizontal position. *Organic murmurs* are usually the result of endocarditis, most often affecting the mitral orifice, systolic in point of time, and with greatest apical intensity. *Palpitation* and cardiac or *precordial pain* are, on the whole, exceptional. An *altered rhythm* is a very common observation in chorea. This has been attributed to chorea affecting the heart-muscle, but is more reasonably referable to functional disturbance and respiratory irregularities, which are very frequent in this disease. Very often the frequency of the heart-beats is the same in both the horizontal and vertical positions. A rapid heart sometimes persists after the attack. Ordinarily, the endocarditis develops during the evolution of the chorea, and is sometimes attended by joint symptoms. Organic heart disease seems to be most frequent from five to ten years of age, but this corresponds to the period of the greatest frequency of chorea. The customary post-mortem findings have been already noted. Of equal or greater importance are the postchoreic observations. Thus, Mackenzie found 66.6 per cent. of thirty-one cases of chorea examined from one to five years after the attack marked by organic cardiac lesions. Osler, in a more extended and very closely scrutinized series, found $51\frac{3}{7}$ per cent. of postchoreic cardiopaths. The same thing is shown by the long-recognized fact of an increasing proportion of cardiac diseases in the subjects of repeated attacks of chorea. The practical deduction points the need of systematically watching the heart throughout the course of, and for a long time subsequent to, the attack of chorea.

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[1] "Il Policlinico," Nov. 21, 1903.

The general state in chorea is commonly reduced. Often, but by no means always, there is some degree of physical depression previous to the onset. Anemia, loss of appetite, gastric indigestion, sluggishness of the bowels, and dryness of the skin are usually developed early in the choreic attack, and are sometimes induced or increased by the injudicious use of arsenic. Ordinarily, chorea is afebrile. In the choreic status and in cases of mixed infection or concomitant sepsis, the temperature rises and is proportionate to its cause.

Course.—The course of chorea, whether of insidious or of abrupt onset, is usually marked by *exacerbations* and *remissions* of the peculiar movements. In the majority of cases, after reaching various grades of severity, the chorea *gradually declines*, leaving the lower extremities, the upper extremities, the trunk, the face, and the tongue, usually in the order mentioned. At a time when the movements no longer occur spontaneously they may still be provoked in the face, and especially in the tongue, by directing it to be vigorously protruded. The average *duration* of the disease is from six weeks to four months, but cases lasting two weeks or less and others lasting six to eighteen months are not very rare. The common *termination* of chorea is in complete recovery, though death occurs in rare instances. A fatal termination results in the very severe cases in which the choreic status exhausts the patient, or more frequently from complications, especially on the part of the heart. Cerebral hemorrhage and softening and concurrent infections may lead to a fatal termination. In other and also very exceptional cases the chorea becomes *chronic*. It may also be followed by a habit spasm or tic.

Recurrence in chorea is so common that it is always to be expected, and occurs in over one-third of all cases. Up to the third attack both sexes show their usual relative susceptibility, but beyond that number the proportion of girls and women rapidly advances in the lists. It is rare to find a male presenting more than 3 attacks, but 10, 12, and more attacks in females are not very rare. Some patients present chorea every spring for several years. As a rule, succeeding attacks grow progressively shorter. Sée gives the average duration as 139, 80, and 55 days in the first three attacks, respectively. It is to be doubted that some of the alleged repeated attacks are such in fact. In many instances a close examination of the patient in the interval will enable the observer to evoke choreic traces in the face, tongue, and extremities, though parents, teachers, and patients insist that complete recovery has taken place. In such instances a recurrence, strictly speaking, is an exacerbation. Choreic girls are also later liable to gestational chorea.

Forms.—Sydenham's chorea presents several forms or modifications that require individual mention. We may distinguish the common form, which has been the basis of the above description; the grave form, the gestational and the paralytic forms.

The grave form, chorea gravis, is marked by an intensification of all the motor and usually of the mental manifestations of ordinary chorea. It is ordinarily of acute and intense onset, or may occur as a sudden recurrence or intense exacerbation of the common type. The choreic movements are wide-spread, continuous, and violent. They may disturb sleep, or prevent it altogether. The patient usually is unable to stand or to sit on a chair, and may be so violently agitated as to be thrown about and off the bed. Swallowing and speech are interfered with or rendered impossible. Excoriations, bruises, and other injuries result. Infections may thus occur, inducing suppurations and erysipelas. Fever arises; there is delirium, sometimes of maniacal intensity. Such a continued, exalted, intensified, chorea has been denominated the *choreic status*. The choreic state, thus constituted, persists for days, and finally subsides, death often following coma. It differs only in degree from the milder attacks, and all gradations may be encountered.

The chorea of pregnancy, chorea gravidarum, usually appears in young primiparæ, commonly during the first half of pregnancy, and is frequently preceded by a history of chorea in earlier life. The predisposing and exciting causes of the common form also obtain here, and the symptomatology is much the same. Cardiac complications are the rule. The motor agitation is usually intense, and there is commonly involvement of the pharyngeal and respiratory apparatus, less often of the laryngeal mechanism. Abdominal and vaginal palpation usually intensify the choreic trouble, and the fetal movements sometimes have the same result. Mental disturbance and affective changes are nearly always present, and tend to persist after parturition or may only develop post partum. Parturition may diminish, augment, or fail to affect the chorea, but, on the other hand, the chorea may cause premature birth and abortion. Ordinarily, chorea, once established, persists until the womb is evacuated and may continue during lactation. It is much more serious than the ordinary form, and terminates fatally in about twenty per cent. of all cases. In some patients it recurs at each subsequent pregnancy.

Paralytic chorea, called *limp chorea* by English writers, is probably more frequent than reports would indicate. The paretic element in chorea has been particularly insisted upon. In this form it is the dominant condition, and the choreic movements are insignificant. It has a predilection for young children, and is most frequent at about six or seven years of age. Commonly, some infectious malady seems to act as a provoking cause or complication. The premonitory stage corresponds to that of the ordinary type. Ordinarily, the paresis appears early and may or may not be preceded by choreic twitching. A monoplegic, hemiplegic, or paraplegic distribution may be presented, but monoparesis is most common. The muscles are toneless and the reflexes abolished. There is, however, no reaction of degeneration, and the outcome is usually complete recovery within a few weeks or months. Muscular atrophy sometimes is noted. It is not unlikely that some cases brought under this head really belong to the neuritides, or to myelitis, or are combinations of these with chorea.

Diagnosis.—The diagnosis of chorea is extremely simple when the motor symptoms are once developed. Difficulty arises usually through mistaking symptomatic choreoid movements in other diseases for true chorea. The escort of attending symptoms should differentiate these pseudochoreas. Here may be mentioned the *rhythmic tremblings* of metallic and toxic poisoning, of hysteria, and of multiple sclerosis. *Friedreich's ataxia, athetosis, and posthemiplegic chorea* have their cerebral signs. *Tics* are more sudden and more completely expressional or gesticulatory in character, showing their subconscious, purposive basis. *Huntingdon's chorea* almost invariably has a familial history, comes on after adult years, and is attended by progressive dementia. The *myoclonias* and the so-called *electrical choreas* present only a superficial resemblance to the chorea of Sydenham. The premonitory symptoms for a given case having been made out, their reappearance often enables the early diagnosis of recurrent attacks before motor symptoms attain prominence.

Prognosis.—In the great majority of cases chorea may be considered a self-limited disease of favorable prognosis. Complete recovery is the rule in children under ten years of age. In proportion as puberty is reached and adult years attained, the prognosis becomes more guarded both as to immediate results and the establishment of chronicity. Recurrent attacks, however, have a progressive tendency to earlier recovery. In a given case the intensity of the attack, the violence of the choreic motions, their generalization, the evidence of degeneracy in the individual, and a bad general physical state unfavorably modify the outlook for early recovery. The prognosis is also affected by the presence or development of cardiac involvement or the appearance of septic processes and high temperatures. The choreic status is of very grave significance, and usually terminates in death. Chorea in pregnancy also furnishes a high mortality for the mother and more often destroys the fetus.

Treatment.—In the treatment of chorea the constant evidence of muscular weakness and mental enfeeblement calls in decided tones for *rest*. Any emotional disturbance promptly aggravates the choreic movements. The child in ordinary cases must be taken from school and kept apart from the romps and often from the gibes of its playmates, and for the most part in bed. A little *chloral* to induce sleep, and frequent sponge-baths, gentle massage, and pleasant, quiet diversion will often, with rest, work great improvement in a few days. *Tonics*, especially *iron*, are usually indicated, and attention to the constipation is always in order. The *diet* should be nutritious and easily assimilated, and contain plenty of fat in the form of cream and butter. It should be carefully controlled, as choreics are very prone to crave indigestible and objectionable articles. The use of drugs is of secondary importance. *Arsenic*, once in vogue, is valuable as a tonic in small doses, but usually harmful in large ones and only rarely efficacious against the chorea when pushed to the utmost limit of toleration. It is capable of producing all sorts of intestinal disturbance, and its protracted use may induce pigmentary changes in the skin or cause a multiple peripheral neuritis. It must, however, be admitted that in some cases a short, vigorous course of arsenic reaching ten to fifteen drops of Fowler's solution, three times daily, for a child six or seven years old, sometimes acts favorably. It may be commenced with a single drop and increased one drop a dose until the stomach rebels, which is usually at about twelve drops. It should then be stopped for a day and renewed at the final dose and slowly increased. It can rarely be used more than three weeks at a time with advantage. The use of *quinin* in large doses, as suggested by Wood, has in a series of cases in the writer's clinic failed to prove of any value.

In the *severe cases* absolute quiet and rest in bed or in a padded corner on the floor is required. Food must sometimes be given by the nasal or rectal tube and chloral must be used freely, associated with bromid if there is much delirium. Even morphin may be required, and strychnin to maintain the heart. Sulphonal, trional, exalgin, and antipyrin have given help in some such cases. Small doses of apomorphin from the one two-hundredth to the one-twentieth by mouth or hypodermatic method, depending upon the severity of the case and the age of the patient, have been strongly recommended.[1] Hot baths and cold packs often are of distinct service. The conservation of strength and the support of the physical forces require most careful thought.

In the *chorea of pregnancy* it is seldom needful to terminate gestation, but if the motor storm is very violent and the mental features pronounced, or the physical state low, it may be indicated, especially as amelioration sometimes follows spontaneous abortion or delivery at term.

Complications such as phlegmons, joint disease, and cardiac involvement must be met on their indications. *Recurrences* should be anticipated and the parents should be taught the significance of sleeplessness, irritability, fidgetiness, capriciousness of conduct and appetite, and at once resume proper treatment. In girls this is particularly needed. In the pubescent period and later gestational experience, watchfulness should be exercised to maintain both the general health and the body-weight at a proper level.

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[1] Tull, "N. Y. Med. Jour.," Mar. 11, 1905.

CHAPTER III.

MOTOR NEUROSES.

THE neuroses brought under the above heading have, in common, a preponderance of motor symptoms. Their grouping is entirely arbitrary and one of convenience.

HUNTINGDON'S DISEASE.

In 1872 Huntingdon called marked attention to a number of families living in southeastern New York, who had for many years been under the continuous observation of his father and grandfather, both medical men. These people were afflicted with a family disease locally known as "megrims" or "megrums," and owing to their peculiar motor difficulties, the patients were commonly called "shakers." The disease has been recognized in many parts of the world, and is variously denominated *Huntingdon's chorea, chorea of the aged, family chorea, adult hereditary chorea, chronic chorea,* and *chronic progressive chorea.* As it has no well-founded relation to Sydenham's chorea, the term *Huntingdon's disease* is here adopted as open to the least objection. The disorder presents an insidious onset in adult life, a marked hereditary character, and a well-defined tendency to mental deterioration and dementia.

Etiology.—The salient etiological feature of Huntingdon's disease is its *heredity.* It has been traced through five generations, and in a given family marks more victims than any other familial disorder, sometimes affecting as many as half of the entire number. It is transmitted about equally by males and females, but presents the peculiarity of never reappearing after once the hereditary chain is broken. Thus it never skips a generation. In contrast to true chorea it affects the male *sex* in greater proportion. Huet collected a series of cases embracing forty-four men and thirty-six women. It is also at variance with chorea in the factor of *age.* It usually appears from thirty to forty-five, but may develop at any later period. Exceptionally, it has been noted at puberty, never in childhood or infancy. Again, rheumatism in the personal and family antecedents is rare. In some instances fright or other mental trauma has appeared to induce it. Epilepsy is sometimes associated with it.

Morbid Anatomy.—It may be stated that no characteristic changes are recorded in the cases examined post mortem. They are those common to dementia in general. Thus Lannois and Paviot [1] in two cases found meningeal thickening, pachymeningitis, cerebral atrophy and compensatory hydrocephalus. The descending cord-tracts and the anterolateral and cerebellar tracts were slightly sclerotic. The cortical layers presented a round-cell infiltration. C. A. Good[1] and Kattwinkel have found in well-examined cases no evidence of inflammation, but widespread degenerative cellular changes throughout the cortex, most marked, however, in the frontal regions.

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[1] "Arch. de Neurol.," Oct., 1897.

Symptoms.—The *motor symptoms* commonly appear insidiously, but in some cases mental enfeeblement precedes. The face, the speech, or the gait may be first involved. Slow involuntary contractions modify the facial expression, cause a hand to start, a finger to move, or compel the feet to deviate from the intended direction. These motions at first are temporarily controllable, or cease on brief voluntary effort. Later, they are not under such control. The *gait* becomes progressively more erratic and uncertain, until, finally, it closely resembles that of drunkenness with the addition of gesticulatory movements of the arms and of facial contortions. The peculiar gestures, poses, and exaggerations of action in these cases are very prominent and often strangely at variance with the mental emotions actually in play. They are increased by embarrassment and emotion, but lessen in repose and subside during sleep. Though bearing a rough resemblance to the movements of chorea, they are more deliberate, gesticulatory, and of greater range. There is usually some muscular weakness, but no other modification of energy and none of sensation. *Speech* is thickened, drawling, and infiltrated with ha's and hem's, but not staccato or explosive, and may finally become impossible. In advanced cases the patient may become bedridden.

The *mental state* is one of progressive enfeeblement and depression. It is of slow onset and its natural goal is complete dementia. It may precede the motor symptoms, but usually follows them at a varying period. Thereafter the muscular and mental disability increase together to the end of life. A *duration* of ten to thirty years is common and old age is often attained. Recoveries are unknown.

Diagnosis.—Huntingdon's disease encountered in a number of generations can offer no diagnostic difficulty. Originating *de novo*, it must be distinguished from chorea proper, from the *maladie des tics*, from double athetosis, and from the family ataxias. *Chorea* has its early mental symptoms, its cardiac lesions, its tendency to recover, and an absence of extreme mental degradation. *Tics* have a limited distribution, are much quicker in their rhythm or activity, and present no dementia unless occurring in idiocy. *Athetosis* is congenital, infantile, or postparalytic in development. The *family ataxias* have their eye symptoms, lack mental degeneracy, and commonly appear early in life.

Treatment has been futile.

MYOCLONIA.

The term *myoclonus* or *paramyoclonus* has been used to designate involuntary, unsystematized, arrhythmic, quick, muscular contractions, similar to those produced by an electric shock. They may be localized or disseminated and may embrace a muscle, a muscle group, or only a few muscular fibers. Under the general term myoclonia may be embraced the *paramyoclonus multiplex* of Friedreich, the *electric chorea* of Bergeron, Henoch, Paget, and the *fibrillary chorea* of Morvan. *Senile chorea*, so called, is generally but the motor index of cortical degenerative changes. Myoclonia is sometimes combined with epilepsy, furnishing the "association disease" of myoclonus-epilepsy, to be described with epilepsy.

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[1] "Amer. Jour. of Insanity," July, 1900.

Etiology.—Practically nothing is known of the causation of myoclonia. Lundborg[1] has suggested that it bears some relation to the thyroid. Nervous heredity is commonly encountered, the male sex preponderates, and adult age is the usual epoch, though Bergeron's form is most common in children. Overwork, fatigue, hunger, fear, traumatism, and cold have been considered excitants.

The nature of the disease is speculative, but the motor cells in cortex and cord are presumed to be at fault. A case in which muscular atrophy followed is thought to add force to this point of view so far as it relates to the cord. The morbid anatomy is practically unknown. Murri[2] found a chronic localized pachymeningitis with atrophy of the central cortex in one case.

Symptoms.—The *onset* may be sudden, following one of the inciting causes mentioned, or the *motor symptoms* may insidiously develop. These are the essential features of the disease, which lacks sensory, trophic, dynamic, and electrical symptoms but usually presents increased tendon reflexes. The clonic contractions begin ordinarily in the lower extremities, and, as a rule, are bilateral, though not strictly symmetrical. They then involve the upper limbs, but commonly spare the face. The clonic contractions are instantaneous and involuntary, increased by emotion, but subject to some degree of voluntary control. They subside or remain in abeyance during voluntary use of the given muscles, at least for a few minutes. Depending upon their location, extent, and intensity, they may appear only as a contracting muscular bundle, producing a linear elevation of the skin, or may cause a joint, a digit, or an entire extremity to start suddenly. Usually clonic, they may be repeated so rapidly as to produce a tonic, or even tetanic effect. One or all varieties may be observed in the same patient. The contractions are unequal, irregular, and arrhythmic. Sometimes they produce constant agitation; sometimes they come on in little attacks, with varying intervals of quiet. They may reach a rate of 60 to 100 a minute, and, as a rule, are more rapid the smaller the muscle affected. They cease during sleep, but in some instances may rouse the patient in the night. Percussion, pinching, heat or cold, electric shocks, and the emotions tend to augment and recall them. The intellect is unaffected. The face, tongue, and trunk are exceptionally involved. The muscles of organic life and the sphincters escape.

When the disease begins insidiously, the contractions come on during repose and at long intervals, attracting little attention. They progressively increase in extent and vigor, and in a few weeks or months reach the period of complete development, which varies in duration in different cases, but is usually protracted many months. The tendency of the disease is then to subside slowly toward complete recovery, but often with remissions or later recurrence. Most cases finally recover.

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[1] "Hygeia," 1900.

[2] "Arch. Ital. di Biol.," 1901.

Diagnosis.—The diagnosis, owing to the rarity of the affection, is seldom made. *Tics* are likely to be mistaken for myoclonia unless the case is carefully studied. These, however, are almost invariably first unilateral, and have a predilection for the face, whence they usually spread to the neck, arms, etc. They are distinctly purposive in character, and expressive or gesticulatory, demonstrating the subconscious basis on which they develop. Jacksonian *fits* are usually attended by sensory auræ, mental disturbance, and major convulsions at some period of the disorder.

The prognosis is good.

Treatment has appeared to have little effect. The use of galvanism to spine and muscles is favored by some, and motor inhibitors, such as atropin, eserin, valerian, hyoscin, and cocain, have been employed with varying results. Commonly, the general physical condition requires appropriate attention.

DUBINI'S DISEASE.

In 1845 Dubini described a disease occurring in the malarial regions of Italy which he called *electrical chorea*. The name is unfortunate, as the condition is not allied to Sydenham's chorea, nor does it resemble it in any manner. This name has also been applied to several varieties of myclonus, and to some hysterical manifestations, with equal disadvantage. The *morbid anatomy* is not yet determined, though many of these cases have been examined post mortem. In nearly all there is evidence of infection, such as pulmonary and splenic congestion, inflammation of the meninges, increase of cerebrospinal fluid, cerebral congestion, especially at the base, and softened foci in the cortex and great ganglia. In some earlier instances the cerebrospinal axis was reported of normal appearance. This statement may be received with some doubt. The disease has been attributed to everything, from malaria to typhus, and seems to be confined to Italy, and especially to Lombardy.

The onset is abrupt and marked by intense, continuous pains in the head, neck, and sometimes in the lumbar region. Shortly the extremities are seized with short, sharp spasms, recalling the electrical responses, and giving rise to the name "electrical chorea." They usually first appear in the upper extremities, especially in the hands and fingers, sometimes in the face, and are attended by painful sensations in the same locality. Gradually they spread into a hemiplegic or diplegic distribution, which is attained within a week or two. The twitchings occur at somewhat regular intervals, and frequently are accompanied by epileptoid convulsions, without loss of consciousness, which may take place several times in the twenty-four hours, and commonly leave paretic traces behind them. Sensibility is not greatly affected, though sometimes hypersensitiveness may be easily provoked and exalts the motor symptoms. Electrical reactions are said to be normal.

The disease usually grows progressively worse. The muscular spasms become almost continuous, the convulsions are rapidly repeated, and in from ten to one hundred and fifty days the disease ends in death in about ninety per cent. of all cases. The fatal termination is preceded by a condition of continuous epileptoid spasm, a sort of status, followed by coma, relaxation, and fatal exhaustion. Occasionally, there are remissions in the progress of the disease. Treatment has been unavailing.

PARKINSON'S DISEASE, PARALYSIS AGITANS.

In 1817 Parkinson gave a complete clinical description of a rather common disease, which he termed *shaking palsy*. It is generally known as *paralysis agitans*. As the usual weakness may be absent and the tremor may appear only late in the disease, the descriptive name is not always applicable. Generally, it is considered a neurosis, but accumulating material points to an organic basis for the malady, which will probably soon pass into its proper category. The disease is one of late middle life, usually commencing locally, tending to hemiplegic and finally to diplegic distribution, and commonly marked by rigidity, tremor, and weakness of similar extent. Never fatal in itself, it lasts until death.

Etiology.—Parkinson's disease rarely commences before forty or after sixty-five years of *age*. Most commonly it appears at about fifty. The male *sex* furnishes over two-thirds of all cases, and this proportion is uniform for all ages. A neuropathic *heredity* is commonly encountered. In exceptional instances the disease appears in several generations or in collateral family branches. Lundborg [1] has recorded five cases in one family, several other members being affected with myoclonia, both of which diseases he thinks related to disorders of thyroidal action. In another case he saw Parkinson's disease with myxedema. Fraenkel [2] has also called attention to myxedematous areas in paralysis agitans which are suggestive of a glandular factor. Among the alleged *exciting causes*, fear, anxiety, grief, and physical exhaustion have been named. In Paris and Strasburg during the sieges of the Franco-Prussian War and in the American Rebellion numerous cases developed under the combined influences of privation, prolonged anxiety, and sudden fears. Traumatism has appeared to incite it, and in such instances the tremor has sometimes appeared in the limb affected, sometimes even in the part of the limb directly injured. Krafft-Ebing [3] traced the disorder to trauma in 7 out of 110 cases. The possibility of hysteria in such cases is strong. Mental shock can not be excluded in such accidents.

Morbid Anatomy.—Regarding the morbid anatomy of Parkinson's disease there is much diversity of opinion and observation. In typical cases Westphal, Berger, Charcot, and Gowers have found no abnormalities. Others have found changes in the encephalon, cord, and even in the muscles. Dubief and Ketscher have noted retrograde changes in the motor cells of the anterior horns and sclerous changes in the white tracts of the cord, recalling those due to senility. Redlich [4] in seven cases found the cord most affected. Small patches of sclerosis were found, mainly in the posterior columns, but some in the lateral tracts, and most frequently at the level of the cervical and lumbar enlargements. They originated from the vessels, and showed atrophy of nerve-fibers and increase in interstitial tissue. The process was an endo- or peri-arteritis, with extension to the surrounding parts. The cells of the anterior gray and Clarke's columns were almost always pigmented. He recognized the changes as senile in part, but thinks they exceed it in degree. Dana has described a diffuse sclerous myelitis with vascular lesions and cellular atrophy in both cord and cortex. Mendel, Charcot, Bloch, and Marinesco in three cases have found tubercles in the peduncular region, and Brissaud, from a study of pseudobulbar palsies in connection with paralysis agitans, is inclined to locate the lesion in the peduncular territory. The monoplegic and hemiplegic cases indicate cerebral disorder and the involvement of the face- and jaw-muscles is certainly due to supracordal states. Gordinier [1] has collated 24 cases examined by recent methods. In all there was decided uniformity of anatomical findings. These involved blood-vessels, neuroglia, and nerve-cells. There was proliferation of nuclei and thickening of vascular walls, increase of neuroglia about the blood-vessels, and patches of perivascular sclerosis, with pigmentation, degeneration, and atrophy of nerve-cells and nerve-fibers. The spinal cord was most affected, the changes being most marked in the gray matter and posterolateral portions of the lumbar and cervical enlargements, usually decreasing brainward, and sometimes not appearing in the encephalon. Though resembling senile changes, the lesions were more intense and general arteriosclerosis was usually absent. At present we can go no further than the supposition that vascular faults are primary, the neuroglia and cellular structures being secondarily involved in degenerative changes. Fatty changes in the muscles are encountered in very old cases, and sometimes a peripheral nerve degeneration of a slight degree. Schiefferdecker and Schultze [2] find the muscle fibers, fibrillæ, and nerve spindles diseased, but no change in the nerves.

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[1] "Hygeia," 1900.

[2] "Deut. Zeit. f. Nervenh.," April, 1899.

[3] "Wien. klin. Woch.," 1899.

[4] "Centralbl. f. allge. Path.," Nov. 4, 1894.

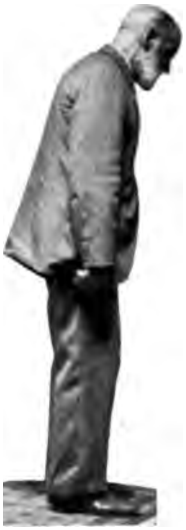

Fig. 234.—Parkinson's disease. Attitude.

Symptoms.—A typical case of Parkinson's disease presents a most striking picture. The patient trots into the room with short, reluctant steps, apparently following his center of gravity. The body is inclined forward, the neck extended and rigid, the elbows flexed and slightly abducted, bringing the hands, with their trembling fingers, to the level of the groins. The face is mask-like, the eyes bright and unwinking. The patient turns bodily, deliberately, and rigidly. He sits down slowly, with precaution, on the edge of the chair, always leaning forward, his shaking hands on his knees in constant motion. Every change of position is studied and reluctant. We may take up the symptoms in detail.

The muscular rigidity is worthy of first attention, as it is the dominant motor phenomenon, causing the peculiar attitudes and postures, the immobile face, and the slowness of movement. It is the motor analogue of the mental inertia so common in this disease. It is nearly always present where the tremor exists, and may be highly developed without the tremor, or may first invade the parts that subsequently tremble. Owing to the rigidity, this reaction-time is increased about forty per cent. Passive motion is sometimes impeded by the rigidity, but there is no hypertonus and the tendon reflexes are only slightly increased. A foot-clonus is never encountered. There is a characteristic *facies*. The nasolabial folds and lines of expression tend to disappear. The face becomes smooth. The brow may retain its cross-wrinkles through enforced elevation requisite for forward vision, if the body and head be bowed. The eyes are widely opened and rarely wink. The ocular globes tend to remain fixed so that the patient, in order to change the direction of vision, ordinarily turns bodily with the neck held rigid. Movements of ocular association and accommodation are impeded by this rigidity, and the patient shows little or no facial variation for any of the emotions that may play behind this mask. *Speech* is modified. It becomes monotonous and deliberate. The patient hesitates, but, once started, hurries his sentences and stops abruptly as if relieved to be through. He likes monosyllables and may become extremely taciturn. In some instances there is an excessive secretion of saliva. In others true bulbar symptoms are added with indications of labioglossolaryngeal palsy.[1]

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[1] "Amer. Jour. Med. Sciences," Dec., 1899.

[2] "Deut. Zeit. f. Nervenheilk.," Dec., 1903.

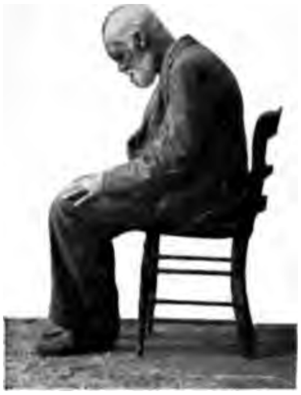

Fig. 235.—Sitting attitude in paralysis agitans.

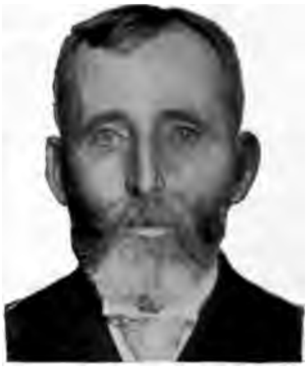

Fig. 236.—Parkinson's disease: facies.

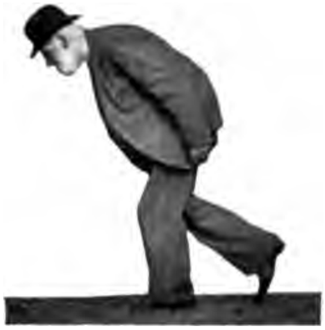

Fig 237.—Gait in paralysis agitans, showing propulsion.

In the *neck* all the muscles are involved, giving an appearance of stiff-neck or rheumatic torticollis, but the face is always held to the middle line, the chin commonly somewhat advanced. The *body* is bent forward throughout its length and also at the hips. There is everywhere a preponderance of flexor positions, as a rule, but in very exceptional cases the neck and body may be bent backward. The *arms*, naturally drooping forward, are flexed at the elbows, which are slightly separated from the sides. The *hands* may be partly flexed or extended at the wrist, but the *fingers* are always held more or less in flexion. A position similar to that of holding a pen is common, or the hand may be partially closed. The digits frequently deviate to the ulnar side of the hand, as in rheumatoid conditions, and these may also be present. In the *lower extremity* the stiffness is less marked, but in advanced cases causes a knee-sprung attitude and gait.

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[1] Bruns, "Neurolog. Centralbl.," Nov. 1, 1904.

The *gait* of Parkinsonians is strikingly peculiar. When the patient rises from the chair, he hesitates a moment as if to take aim, and starts ahead in a direct line, his laggard legs trotting to keep up with the forward-leaning body. In some instances the patient is strongly impelled forward, and can only arrest himself by running into objects or passers-by. There may or may not be an actual tendency to fall forward, or *propulsion*, but in some cases, if the body be started backward, sideways, or forward by a push, the direction is maintained for a few or many steps, giving rise to the terms *lateropulsion* and *retropulsion*, and these may occur spontaneously on getting up or in attempting to stop while advancing. As described by Stewart,[1] in advanced cases the method of *getting into bed* is characteristic. The patient climbs on to the bed, stands up, and, bending down very slowly, grasps the rail at the footboard. Holding firmly to the bedstead he slowly sits down and then falls or rolls backward into the recumbent posture.

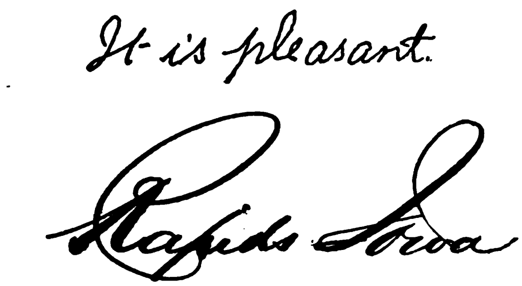
It is pleasant.

Fig. 238.—Handwriting in Parkinson's disease, with former style below.

The trembling in shaking palsy may appear after rigidity has developed or at the same time. It usually commences in one hand and arm and then invades the lower extremity, subsequently appearing in the opposite arm and finally in the opposite leg. In some cases it is bilateral from the start, but commonly it is more marked on one side than on the other, and may be monoplegic or more often hemiplegic for several years, eventually showing a tendency to diplegic distribution. Often it is steadily preceded in its advance by the rigidity, or the rigidity may be generalized and of long standing before tremor appears in the hands. It affects the distal portion of the extremities most. In the *hands*, where it customarily first appears and is most developed, it causes a rhythmical, alternating flexion and extension of the fingers, mainly at the metacarpal joints. The tremor may be limited to the index and thumb, or affect the interossei most, causing rolling of the fingers upon their long axes. The patient appears to be constantly rolling some small object, as a pill or a pencil, between his fingers and the opposed thumb. Sometimes flexion and extension of the wrist are added, and very rarely we encounter movements of pronation and supination. As a rule, the arm and shoulders are unaffected. In the *lower extremity* the tremor predominates at the ankle, causing the foot to drum on the floor as if with clonus. The toes are less evidently involved by tremor, but many patients complain of flexor cramping of the toes as a very early condition. This usually comes on while walking, and may bring the patient to a momentary standstill. The muscles of the thigh often participate in the tremor. The muscles on the back of the neck, shoulders, and dorsum of the body are least affected. The abdominal muscles apparently escape. In the great majority of cases the *head* does not tremble, or only does so by movements communicated to it from a distance. In very rare cases, however, there is a rhythmic nodding, shaking, or rotation of the head that may persist even when the patient is recumbent. The *eyelids* exceptionally are affected, while the *lips* and *lower jaw* not rarely show a tremor synchronous with that in the hands.

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[1] "Lancet," Nov. 12, 1898.

The great peculiarity of this tremor is its usual occurrence while the patient is *at rest*, during repose, and while the parts are supported. In a minority of cases, however, and in other cases at an early period, the tremor is *intentional*—that is, analogous to the tremor of multiple sclerosis. The tremor ceases during sleep, and usually it subsides momentarily on voluntary motion. In early cases, and particularly in cases presenting marked antecedent rigidity, the tremor may only appear on voluntary and sustained motion, as in reaching to the back of the neck or into a distant pocket, and must be carefully sought. The tremor is a slow one, of from four to eight oscillations in a second. Usually the movements are more rapid if of limited extent, as when confined to the fingers, and grow slower as they involve larger muscles. When both upper or all four extremities are involved by the tremor, there is a practical *synchronous uniformity* of rhythm at all points. The character of the tremor shows distinctly in the *handwriting*. The letters are formed slowly and are of fair proportions, but all the lines are tremulous, both upstrokes and stems. The writing tends to become cramped and small. Sometimes a lens is required to detect the tremor thus graphically demonstrated.

The palsy never reaches a complete degree, and the paresis may be extremely slight. While patients may bitterly complain of a feeling of weakness and stiffness, they often show a normal amount of strength, even when the rigidity and tremor are very well developed. In advanced cases, however, there is customarily some weakness, and this may even be extreme. These patients, as a rule, are loath to make exertion of any sort.

Sensory Disturbances.—The general sensibility is practically objectively intact, but Palmieri and Arnaud [1] insist that the parts affected by tremor show decided hypalgesia, most intense in the distal portions of the limbs and gradually shading off toward the trunk. Karplus,[2] in a study of 103 cases, never found objectively disturbed sensation without tremor, and subjective sensory complaints were noted in but 35 per cent. Parkinsonians frequently complain of subjective *feelings of heat*, more rarely of *cold*, and often of dull aches and indescribable discomfort in the affected limb. It is not rare for Parkinsonians to complain of considerable pain in the early stages of the disease similar to rheumatic pains in joints and muscles. The heat sensations may be accompanied by *vasomotor disturbance*, showing itself in elevated local temperature, in profuse sweats, and in flushing. Sometimes patients seek cool rooms and throw off heavy clothing and bed-covering, even in winter. In the majority of cases complaining of heat the surface temperature is actually increased. In some cases areas of *brawny skin* are encountered on the brow or body, a condition suggestive of myxedema and of scleroderma. Often there is great restlessness, apparently due to the discomfort arising from muscular rigidity. The hands may be frequently moved or the patient insists upon the limbs being rubbed and moved about every few minutes. Muscular atrophy only appears in advanced cases, but even then is not extreme. There are no electrical changes or sphincter weakness. As this is a disease of the involutional period of life, we may find all the disturbances of senility as coincidental accessories.

The mental state is likely to be mistaken for one of dementia, but, as a rule, these patients enjoy all their mental powers. There is, however, the same inertia in the mental processes that marks the muscular state. They shun exertion, are chary of their thoughts, talk little, appear indifferent, and often require the incentive of strange faces or extraordinary circumstances to arouse them to a show of mental activity. This, taken with their inexpressive faces, is easily misleading.

Course.—The disease is essentially chronic and progressive. The *onset*, insidious, as a rule, may be abrupt, following some mental or physical storm. The *duration* is from ten to forty years. In the extremely protracted cases the patients become more and more helpless, fall into a senile dementia, and die from intercurrent disease, usually pneumonia. *Varieties* have been described, but they are usually limited and undeveloped cases. Thus, the monoplegic and hemiplegic forms, the form without tremor, the form without rigidity, and the form showing extension are named. The *prognosis* is bad, but there may be remissions.

Diagnosis.—In typical cases the diagnosis is made at a glance. In early and undeveloped cases Parkinson's disease may be mistaken for *posthemiplegic trembling*, but lacks the history of a stroke. *Senile trembling* usually first affects the head and does not present the facies and rigidities, but intermediate cases may be found and both may coexist. *Multiple sclerosis* has its increased reflexes and intention tremor, *hysteria* its stigmata.

Treatment.—If seen early, the case should be treated as one of cerebral arteriosclerosis (see p. 201). In several instances this plan of treatment has seemed to retard the development of the disease. Symptomatic medication is practically useless. Opium, hyoscin, and cannabis indica, if given freely, temporarily control the tremor, but at the expense of the general health and welfare. Massage, electricity, and strychnin give a little help for the time being in some cases. Persistent exercises,—both passive and active,—Swedish joint-movements, and gentle muscle kneading, if intelligently carried out for a long period of time, certainly benefit these patients in the earlier stages. Mental and physical fatigue must be avoided. Charcot noticed that the vibrations experienced in carriage- and car-riding mitigated the tremor, and treated many cases by means of a jolting or vibrating chair with temporary benefit.

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[1] "Clinica Med. Ital.," No. 6.

[2] "Jahrb. f. Psych. u. Neurol.," 1900.

THOMSEN'S DISEASE (MYOTONIA).

In 1876 Thomsen, himself subject to the disease, fully described a muscular condition later called *myotonia congenita, family myotonia,* etc. It is a disorder manifest in the voluntary muscles, which show a stiffness and rigidity upon attempted use after a period of repose, and certain peculiarities of mechanical and electrical irritability. It is a rare disease, numbering not more than a hundred recorded cases.

Etiology.—The salient etiological feature of the infirmity is heredity. Most of the known cases have been in family groups, sometimes extending over several generations and through several collateral branches. Occasionally the disorder has passed over a generation and again appeared. With and without direct heredity the family history is commonly surcharged with neuroses and psychoses. Males are apparently the more commonly affected. Von Bechterew [1] suggests that a self-poisoning or auto-intoxication is active in this condition, and Jacoby [2] looks upon the disease "as due to an embryonal developmental disorder of the nerve-cells, consisting in the more or less diminished resistance of the cells to the influence of toxic processes." The disease is also related to the myopathies, as shown by the muscular contours and the occasional appearance of atrophy.[3]

Morbid Anatomy.—Various observers, upon examination of excised fragments of the affected muscles, have recognized a hypertrophy of the protoplasm and nuclei and a deficiency of striation in the enlarged muscle-fibers, usually with slight but insignificant increase of interstitial tissue. This constitutes, according to Deleage, a persistence of embryonal conditions. Jacoby insists that this appearance is an artifact that is not found if the tissue removed during life is not allowed to contract. Babes and Marinesco have noted deformity or maldevelopment of the terminal motor-nerve plaques. Schiefferdecker asserts that there is a distinct disorder of the sarcoplasm and disease of the muscle fibrils. The hypertrophy appears to be secondary and the increase of nuclei is proportional to it. In the only autopsy yet recorded, Déjerine and Sottas[4] found no changes in medulla, cord, or peripheral nerves. It is as yet impossible to say whether we have to deal with a pure myopathy, a trophoneurosis, or a congenital defect in the trophic and motor apparatus of the cord.

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[1] "Neurol. Centralbl." Feb., 1900. [2] "Jour. Nerv. and Ment. Dis.," July, 1898.
[3] Bernhardt, "Allg. med. Centralzeit.," No. 14, 1890. [4] "Rev. de Méd.," Mar., 1893.

Symptoms.—The awkwardness caused by the fixity of the muscles upon attempted use is noticed in infancy, or may appear at any time up to the twentieth year, or perhaps even later. In typical cases, when the subject desires to execute some movement, a more or less marked and prolonged contraction fixes the muscles in question. This gradually subsides and the movement is accomplished. Repetitions of the spasms occur progressively with less force and duration for the particular action, and finally cease to appear, but any change in the character or even in the rhythm of the movements may reinstate the muscular fixation. Thus, in rising from a chair the legs and thighs are held rigidly. Once erect, the first step is impeded, the second less so, and finally steps are taken with natural ease, but a halt, a sharp turn, or even a change of speed may again set up the spasm. All the voluntary muscles may be affected, even to those of the thorax, eye, and tongue, but usually the myotonia is most marked in the lower extremities, and in some cases the face and upper extremities escape. The *congenital paramyotonia* of Eulenberg, in which symmetrical groups of muscles are affected mainly on direct exposure to cold, appears to be a limited form of Thomsen's disease. Both varieties have been observed in the same patient by Bernhardt.[1] The sphincters and unstriped muscles escape, and in the case mentioned by Eulenberg the heart-muscle was normal. Gaping, sneezing, cold, wet, fatigue, and emotional excitement provoke the spasms, while warmth, moderate exercise, repose, and quietude diminish their intensity. They are in some measure relative in intensity to the vigor of the attempted movement.

Commonly, the affected muscles are of unusual firmness and increased bulk, but of lessened power, giving an appearance of athletic development at variance with the actual weakness, in many cases suggesting a pseudohypertrophy. They are perfectly supple to passive movements. The reflexes are normal, but a tap on the tendon is likely to produce a spasm in the anterior femoral group, modifying the usual response. Electrical and mechanical excitations of the motor *nerve-trunks* produce perfectly normal responses, or they are, if anything, somewhat diminished. In the *muscles* it is very different. A slight blow, as with a percussion hammer, produces a persisting welt from localized muscular swelling or myoidema. The galvanic current produces sluggish, prolonged contractures upon closure almost equally with either pole. Anodal or cathodal closing tetanus may often be secured with the continuous passage of five to ten milliamperes of current, and anodal opening tetanus is not infrequently observed. The strong faradic current produces undulatory contractions in many muscles, and these sometimes attend the passage of the continuous current. Repeated electrical or mechanical stimulation of the muscles, like volitional use, gradually exhausts the myotonic responses.

As *varieties*, Jacoby would limit the term congenital myotonia to cases (1) presenting a hereditary etiology either as a direct transfer from the ascendant, or by inherited disposition; (2) manifesting the myotonic disorder of movement—namely, intention spasm; (3) showing the myotonic reaction, which he describes as made up of normal mechanical and faradic excitability of the nerves and increased mechanical and faradic excitability of the muscles, anodal and cathodal contractions being equal and the response always being tonic and prolonged; (4) persisting hypertrophy of the enlarged muscles; and (5) absence of symptoms pointing to gross involvement of the nervous system. Other varieties of myotonia he would denominate myotonia aquisita, as describing those acquired subsequent to birth, and myotonia transitoria for the cases due to exposure to cold, etc. Lannois[1] has noted an association of progressive muscular atrophy and myotonia in an individual of apparently excessive muscular endowment and finds that about a dozen such cases have been reported.

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[1] *Loc. cit.*

Psychic disorders are frequently associated, but not necessarily present. The malady, once developed, tends to persist for life, which it does not abridge. It is an incurable infirmity, but sometimes shows arrest or amelioration.

Diagnosis.—The myoidema and myotonic electrical reactions, taken with the intention spasm, if the term may be used, make the diagnosis easy. *Tetany* has its distinctive signs in the phenomena of Trousseau and Chvostek. *Pseudohypertrophic paralysis* has peculiar deformities, contractures, and weakness without intention cramps. It subsequently shows atrophies and has no myotonic reactions.

Treatment.—The causes which provoke the myotonic cramps, such as overexertion, fatigue, exposure to cold, and excitement of all sorts, must be avoided. Massage, reasonable exercise, electric baths, and cerebral galvanization have been recommended.[2] A careful search for toxic factors should be made, and conditions known to be attended by them should be corrected.

FAMILY PERIODIC PARALYSIS.

There is a form of periodic limp paraplegia that may be designated *family periodic paralysis*. It is characterized typically by recurring attacks of pure flaccid motor palsy, most pronounced in the lower extremities, marked by diminished reflexes and lessened electrical and mechanical muscular excitability. The intervals are those of ordinary health, and there is a decided family and hereditary tendency.

The first definite outline of this condition was given by Westphal, in 1885. Additional material has been contributed by Goldflam, Oppenheim, Bernhardt, Hirschl, and other German observers, and by Burr, Taylor, Mitchell, Putnam, and Crafts in this country. Oddo and Audibert[3] were able to collect 64 published cases.

Etiology.—*Heredity* is most apparent. Taylor noted that thirty-five cases had been reported in three families, nineteen in one of them, and in two instances it had appeared in five consecutive generations, descending through *both sexes*, which are about equally represented. Nearly all known cases presented attacks before the *age* of twenty-five, the great majority in youth, rarely, however, before the tenth year of life. The *inciting causes* of the attacks have been exertion, fatigue, and mental strains. Goldflam, in 1890, suggested that there was an *autotoxic* causation acting upon an inherited vulnerability of motor nerve-cells, and Crafts [1] and Irwin have been able to isolate an extractive from the feces passed immediately after an attack which produced temporary paralysis when injected into rabbits and guinea-pigs. The amount of urea excreted during the attack also appears to be diminished. These results have not been confirmed.[2] Somewhat analogous attacks due to malarial infection have been cured by quinin. The muscular contours, and in Bernhardt's case atrophy of the thenar eminences and continued muscular weakness, imply a relationship to the progressive muscular atrophies and to the myotonias. Examination of muscular fibers taken from the living subject also indicates a similar kindred.

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[1] "Nouv. Icon. de la Salpêt.," Nov., 1904.

[2] W. von Bechterew, "Neurolog. Centralbl.," Nov., 1897.

[3] "Arch. gén. de Méd.," 1902.

Symptoms.—The *attacks* come on rather slowly within a few hours, usually at night, or during sleep. They are sometimes preceded by a feeling of weariness, numbness, formication, sweating, heat, desire to urinate, headache, backache, rapid pulse, coldness of the legs, etc., in various cases, but all prodromata may be lacking. In some instances the attacks begin as a migraine or alternate with migraine.[3] The *lower extremities* are uniformly most affected, and the paralysis may be limited to them, but in other instances involves every skeletal muscle except those controlled by the cranial nerves, so that the patient is inert from the chin downward. A facial weakness has been observed in one case. Even the voice and respiratory efforts may be weak, sneezing and coughing impossible, and the heart has in certain instances been found dilated and with evidence of mitral insufficiency, both disappearing with the attack. Such attacks last from an hour to a week and tend to uniformity in a given case. The paralysis recedes in the reverse order of invasion. Attacks may *recur* daily, weekly, or once in several years.

During the attack *electrical responses* in nerve and muscle and the tendon *reflexes* are lessened or completely abolished, but there is neither reaction of degeneration nor sphincteric incompetence. *Mechanical stimulability* of the muscles and nerves also disappears. The mind and general sensation and the special senses are unimpaired. The attack subsides about as rapidly as it develops, and in the intervals the health is usually perfect. Indeed, many of these patients are described as unusually robust and commonly very muscular. The muscular contours at times have even suggested the myopathic family disorders.

Diagnosis.—The diagnosis in familial cases should be easily made. In sporadic instances the behavior of the reflexes, electrical and mechanical responses, periodic recurrence and absence of mental and sensory disturbances, should be sufficiently definite to distinguish the condition from *hysteria*, with which it has been confounded. A first attack may suggest *Landry's paralysis*, but again reliance may be placed on the quantitative electrical change.

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[1] "Am. Jour. Med. Sciences," June, 1900.

[2] Mitchell, Flexner, Edsall, "Brain," 1902. Singer, "Brain," 1901.

[3] Holtzapple, "Am. Med.," April 30, 1904.

Prognosis and Treatment.—Thus far no treatment has been of much service, but those conditions, such as fatigue, which appear capable of inducing attacks in given instances must be avoided. In the migrainous cases bromid and caffein have proven useful. The tendency is for the disease to endure for life without compromising it. A further knowledge of the toxic states may furnish the key both to the pathology and treatment of this rare disease.

FAMILY TREMOR.

A tremulous condition, particularly of the hands, but also in some instances involving the head, face, and tongue, closely resembling the tremors of multiple sclerosis, senility, alcohol, and mercury, is encountered as a family trait. It may be traced through several or many generations, and affects a large proportion of the family members, appearing before forty and usually before twenty years of age.

The majority of such families are markedly neurotic, and, according to Raymond, the tremor may be considered as a stigma of degeneracy. In character the tremor is usually fine and rapid. It is accentuated by effort, fatigue, and emotion, ordinarily does not occur during rest, but generally closely resembles the intention tremor of multiple sclerosis.

CHAPTER IV.

FATIGUE NEUROSES.

MANY occupations requiring the constant repetition of certain precise muscular movements may, eventually, through overuse and fatigue, give rise to disturbances of muscular control for the maneuver in question. The condition may be manifest as pain, tremor, weakness, or cramp, but usually these are variously combined in different cases. This group of motor disturbances is also called *occupation spasms* or *occupation neuroses*. Many of them are described under terms indicating their particular avocational association, as scriveners' palsy, piano-players' cramp, seamstresses' spasm, etc. We may take writers' cramp, the most commonly encountered one, as a type, and then only an enumeration of the other forms will be needed. The more delicate and highly differentiated the functional movements, the more readily does their repeated overfatigue set up this inhibitory condition. No definite anatomical changes have been found, but it is highly probable that improved methods will demonstrate morphological alteration of the nuclear gray. Vigouroux[1] claims to have found changes in nerves and muscles in many cases, and supposes them to be present in all.

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[1] "Progrès Médicale," quoted in "Amer. Medico-Surg. Bulletin," Jan. 25, 1897.

WRITERS' CRAMP.

Writers' cramp is variously known as scriveners' palsy, graphospasmus, mogigraphia, chirospasm, etc.

Etiology.—A *neuropathic heredity* and a neurotic make-up are very common among sufferers from writers' cramp. The neurosis has exceptionally been noted in brothers and in parents and children, but, ordinarily, there is merely a transmitted nervous tare constituting a tendency to the development of the disorder under the provocation of overuse of a certain functional group of muscular movements. It is much more common in the male than in the female *sex*, perhaps owing to the greater proportion of men engaged with the pen. It most commonly develops between twenty and fifty years of *age*, being very rare before and after these extremes. Its greatest incidence is between twenty-five and thirty-five. It is very likely to appear during periods of physical or mental strain, especially after protracted anxiety arising from any cause. Occasionally, some *local injury* to the hand or arm which entails additional difficulty in the mechanical process of writing may serve to provoke it. Neuritis, neuralgia, and hemiplegia affecting the writing arm have been followed by the neurosis. The principal inciting cause of the trouble is *excessive writing in a bad manner*—that is, in any style that depends upon the employment of the small muscles of the hand, wrist, or forearm in chief, and in which the writing movements are not made entirely from the shoulder. Thus, writers' cramp is practically unknown among stenographers, in spite of the rapidity and tediousness of their work, owing to the fact that the characters employed are best made by the freehand method customarily employed. Gowers, indeed, encountered a stenographer who could write shorthand readily, while ordinary script caused a spasm.

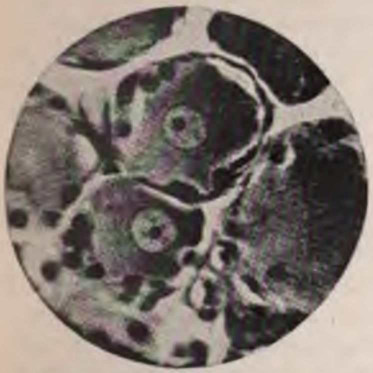


Fig. 239.—Unstimulated cell from posterior spinal ganglion of cat (from Hodge, after Tuke).

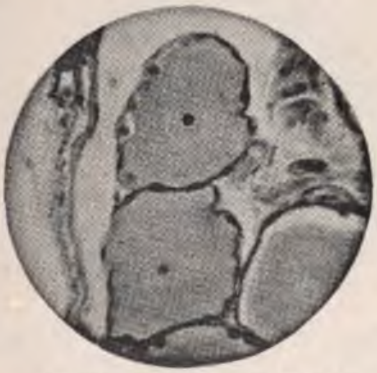


Fig. 240.—Cell from posterior spinal ganglion of cat. First effect of stimulation (from Hodge, after Tuke).

Pathology.—Numerous theories have been advanced regarding the pathology, and the seat of the disease has been variously placed in the muscles, in the nerves, in the spinal centers, in the cerebellum, and in the cortex. We can, at once, rule out the muscle and the nerve as initial loci, because in ordinary cases the muscular control for movements not of the particular occupational variety are fully, promptly, and normally executed. It is impossible to conceive of a peripheral lesion which would disturb only a certain purposive function, leaving others intact. In most all cases the motor function at fault is one to which the motor apparatus has become trained and habituated. In some degree it is automatic and subconscious. Such acts are generally supposed to be largely subcortical. Another factor is interposed by the fatigue element. Hodge has clearly demonstrated the changes in motor cells resulting from physiological fatigue. In the occupation neuroses it is at least supposable that the fatigue may overpass the limit of recuperation. Well-authenticated cases in which muscular atrophy has succeeded the spasm would indicate rather conclusively that such was the case, that the motor cells of the cord were at fault, and that their trophic powers were finally involved. Sensory and motor symptoms, functionally grouped, would also point to the cord, but it is impossible as yet to exclude the superior cortical centers.

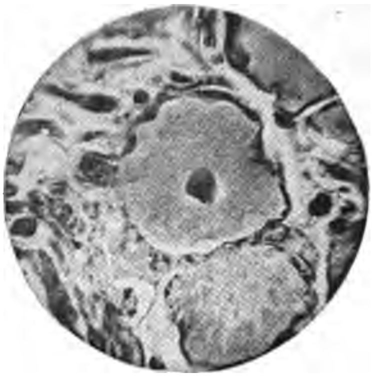


Fig. 241.—Cell from posterior spinal ganglion of cat. Later effect of stimulation (from Hodge, after Tuke).

Symptoms.—The *onset* of writers' cramp is commonly insidious. It is noticed after writing a considerable time that there is sensory discomfort or motor difficulty. The hand aches, feels numb or weak, or may become cramped or tremulous. After a moment's rest and a little rubbing writing may be resumed for a time, when the trouble reappears and gradually less and less work is required to induce the disabling condition, until in extreme cases merely putting the hand in the writing position may cause it. Several forms have been described depending upon the major symptom. Thus we have the *spastic, tremulous, neuralgic*, and *paralytic varieties;* but they are very rarely pure, most cases showing a combination of sensory and motor features. Ordinarily, the cramp is painful not from the muscular contraction, but because the parts are affected with neuralgic pains that are occasioned only by the act of writing. In some instances they are so severe as to alone attract the patient's attention and inhibit the act of writing. In other and much rarer cases a feeling of weakness appears to prevent the use of the pen, but it is usually associated with pain. Tremor may be the principal feature, and is practically a manifestation of weakness.

Motor Disorders.—In the case, Fig. 242, illustrated from photographs, the plan of holding the pen between the index and middle fingers was adopted when spasm first developed, and for a time permitted a continuation of clerical work. When seen later, the hand would begin to cramp after half a dozen words were written, as shown in the second cut, and in a moment the spasm would become violent, the pen would fall, and the fingers and thumb would be extended as shown in the third cut. This was an aggravated case. Commonly, as soon as the spasm causes uncertainty in writing the act is stopped and the spasm subsides. It may cause a great change in the writing, which tends to become cramped and full of uncertainties and zigzags. The pen is forcibly jerked and thrust in various directions. At first, by steadying the writing hand with the other, and adopting a thick penholder or one fitted with a ball held in the palm of the hand or with rings against which the fingers are pressed in extension, writing may be continued for a time, but usually the spasm reappears. If the other hand be then used, it is not uncommon for the spasm to appear on that side after a few months, though this is not invariably the case. When the spasm affects the second hand, it may also cause the muscles of the first hand to contract. The spasm may also spread to the muscles of the forearm and even to the arm and shoulder.

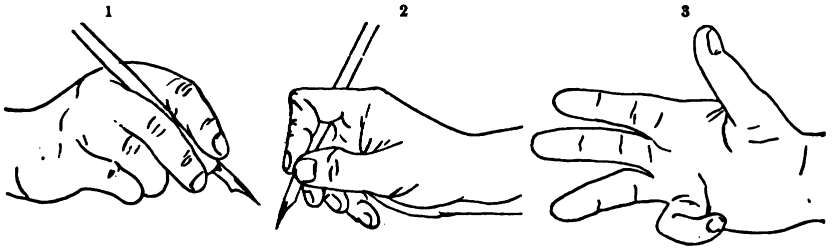
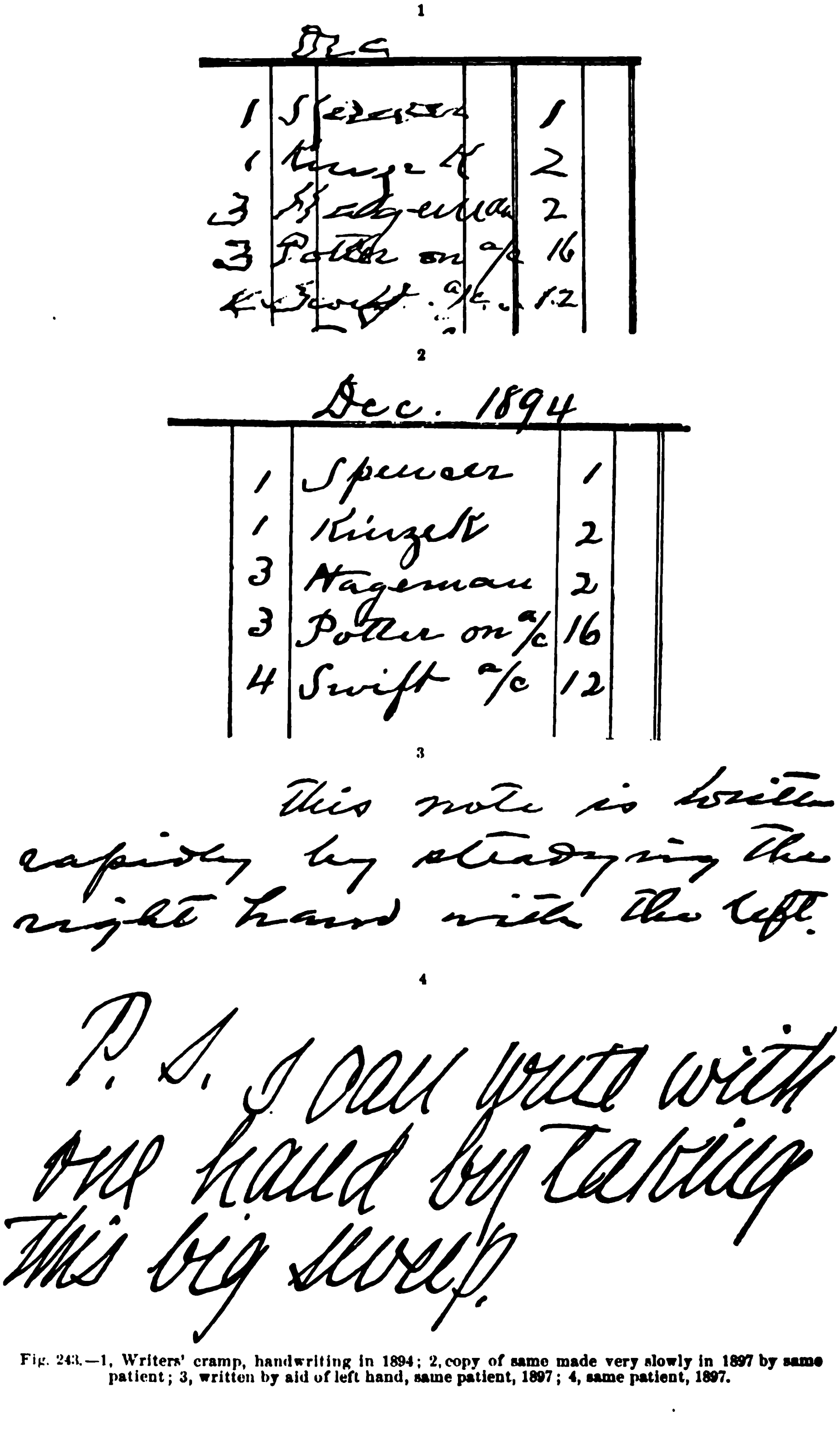


Fig. 242.—Writers' cramp (from photographs): 1, Pen held between fingers at first gave relief; 2, spasm beginning in thumb and index; 3, fully developed spasm, throwing all the digits except smallest finger into extension and causing the pen to fall.

In cases of moderate severity the spasm may be occasioned only by the act of writing, but sometimes, and in severe cases usually, other acts requiring precision in finger movements also call it forth. In telegraphers' cramp, writing with the Morse key or the pen often alike causes the spasm; musicians may experience the spasm both in instrumental manipulations and in writing; seamstresses by using the needle or the scissors, etc.

The power of the hand for coarse muscular efforts may be unimpaired, but in cases of long standing some weakness in the muscles most concerned is commonly found, and in such cases slight wasting may occur. In rare instances, when the use of the affected member is pushed in spite of the disability, permanent atrophy may result. In figure 244 is shown the hand of a seamstress in which local pain and spasm were finally followed by atrophy, the reaction of degeneration, and fibrillary twitching limited mainly to the radial half of the hand and ulnar border of the forearm, namely, to the muscles chiefly concerned in the use of needles and scissors. The electrical responses in muscles and



1

1	Spencer	1
1	Kinzelt	2
3	Hageman	2
3	Potter on a/c	16
4	Swift a/c	12

2

Dec. 1894

1	Spencer	1
1	Kinzelt	2
3	Hageman	2
3	Potter on a/c	16
4	Swift a/c	12

3

This note is written rapidly by steadying the right hand with the left.

4

P. S. I can write with one hand by taking this big scrawl.

Fig. 243.—1, Writers' cramp, handwriting in 1894; 2, copy of same made very slowly in 1897 by same patient; 3, written by aid of left hand, same patient, 1897; 4, same patient, 1897.

nerves may be normal, but in protracted cases there is usually an increase and finally a decrease of excitability, and, as in the rare case illustrated below, the reaction of degeneration may be present.

Sensory Disorders.—The sensory disturbance is sometimes described as one of great fatigue, or there may be pain and tenderness. Tenderness along the course of the nerves is not uncommon. In some cases pain is located in the small bones and joints of the wrist or fingers. It may only occur on attempts to write, and it may or may not be preceded by spasm. In certain cases it extends to the arm, axilla, and shoulder. Such paresthesias as "pins and needles," tingling, ache, "tired feeling," etc., are often complained of, and may similarly extend up the arm. Actual hyperesthesia or anesthesia are encountered only when neuritis is associated.

Course.—Commonly insidious in onset, writers' cramp may develop with some suddenness, after an emotional, moral, or physical storm, and tends to progress so long as writing is persistently attempted. If the opposite hand be trained, it usually also becomes the seat of cramp, which is likely to increase more rapidly than on the side first affected. If writing be completely abjured, the cramp gradually becomes less and less, and may entirely cease after a few months or a year, but is very likely to return if continuous writing is again taken up. The tendency of the cramp, pains, and paresthetic sensations to persist and increase if the writing occupation is continued is pronounced, and there is danger of pushing the condition into one of organic changes, with atrophy and permanent palsies.

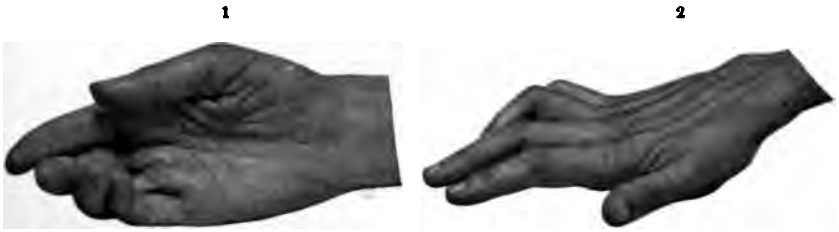


Fig. 244.—Wasting of muscles in a severe case of seamstress' cramp.

Diagnosis.—The diagnosis is usually made by the patient, and nearly every large clerical force and telegraph office furnish cases. *Monoplegia* and *hemiplegia* from organic brain disease are sometimes mistaken, as the patient's attention is first attracted by his clumsiness in writing. *Musculospiral palsy, neuralgias,* and *rheumatic* pains are occasionally mistaken for writers' cramp, but in all these conditions the symptoms are persistent and not alone provoked by writing. Spasm, usually present in writers' cramp from the first, is also absent. *Hysteria* may present limited tremor and motor difficulty, but the disability is not confined to writing, and sensory stigmata are added.

Prognosis.—The prognosis is very unfavorable if writing be persisted in. If the patient can completely abstain from the use of the pen or similar writing implements, the spasm tends to subside and disappear unless the case is of long standing and so aggravated that all sorts of finger use induce the cramp. The neuralgic cases are somewhat more easily managed than the cramp variety if prolonged rest can be secured. The presence of any removable diathetic disability, as lithemia, favorably modifies the outlook, and the termination of general depression, physical or mental, may lead to marked improvement in the spasmodic affection. The tendency to recurrence and to extension has already been noted. It is rare for a patient, even after years of rest, to be able to return to writing occupations without a reappearance of the neurosis.

Treatment.—Recognizing the fatigue element in the production of writers' cramp, *rest* becomes the principal note of treatment. All *physical failings* and conditions which tend to make fatigue of ready appearance must be scrupulously sought and carefully managed. Absolute cessation of writing is of the first importance. Unfortunately, patients whose living depends upon clerical work will not or can not conform to these requirements until the neurosis completely disables them. *Exercises* to develop the strength of the arms, hands, and fingers should be employed, and massage, baths, and electricity, for their general tonic effect, are of some service. When writing is again taken up, it must be by the proper full-arm movement. A typewriting machine can often be substituted for the pen. The use of spinal sedatives to control the cramp and permit a continuance of writing is pernicious, and rarely gives even temporary help. When writing is resumed, the free-hand method from the shoulder must be employed, and the splint (Fig. 245) affords a help by immobilizing the wrist and fingers during writing exercises.

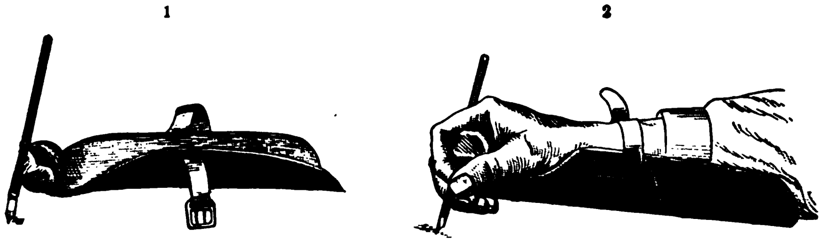


Fig. 245.—1, Splint to immobilize hand and wrist in the treatment of writers' cramp; 2, splint in use.

OTHER OCCUPATION NEUROSES.

The general considerations pertaining to writers' cramp are equally applicable to the other occupation neuroses, and with proper modification the particular features are identical. Among the occupation spasms more commonly encountered are the cramps of violin and pianoforte players, telegraphers' cramp, seamstress' cramp, and hammer cramp in smiths and artisans using the hammer. Artists, flower-makers, turners, watchmakers, knitters, engravers, masons in using the trowel, sailors from pulling on ropes, treadlers, compositors, enamelers, cigarette-makers, shoemakers, milkers, money-counters, letter-sorters, and players on various musical instruments, including drummers, comprise the list given by Gowers. It has been noted in a shoe salesman from the stooping position needed in putting on shoes, in gum-chewers, affecting the masticatory muscles, and in various factory employees who incessantly use the same movement in feeding or attending some machine. Clergymen and other public speakers, from a faulty use of the vocal apparatus, may acquire a laryngeal neurosis that is manifest every time the voice is strained, causing a sudden loss of modulation, which may end in continuous vocal disability. A spasm of a similar nature, involving the lumbar muscles, has been seen in physicians, due to continuous riding in carts or buggies.

CHAPTER V.

NEURASTHENIA.

In 1869 Beard, of New York, directed general attention to a nervous state or condition marked by irritable weakness, and adopted for it the name of neurasthenia. It is familiarly known as *nervous prostration* or *nervous exhaustion*, and is a fatigue neurosis. In Europe it was at first somewhat derisively called the *American disease* or *Beard's disease*. It is now recognized the world over as a morbid state; as one of all time, and not a product of modern life or of American conditions. It is marked by a host of subjective symptoms and a very few objective phenomena, all more or less variable, and most of them inconstant. As a rule, all forms of nervous energy—psychic, motor, and organic—are reduced, so that fatigue is more quickly occasioned than in health. There is less endurance, and consequently greater irritability, which most shows itself in the mental sphere. The whole state is summed up in the classical term "irritable weakness."

Etiology.—Neurasthenia seldom occurs before twenty years of *age* or after fifty. It is essentially a disorder of the energetic, productive, troubled period of life. Both *sexes* suffer, and perhaps in equal proportion, but in females a more common association with hysteria frequently carries these cases into that list. In this country the *high altitudes* of the Western plateaus and the *extremes of climatic conditions* in the Northern, and particularly the Northwestern, States apparently account for the greater frequency of neurasthenia in these localities than on the seaboard and in the Southern States. All *races* present the disorder. Hebrews and Slavs are said to be very subject to it, and Scandinavians, at least in this country, furnish a large contingent. A *neuropathic heredity* is less common than in hysteria or epilepsy, but neurasthenics are very commonly found at the head of a neuropathic strain. *Debilitating conditions in the antecedents* of neurasthenics are very common. Gout, rheumatism, tuberculosis, syphilis, excesses, dissipation, malaria, and all the cachexias in parents are likely to discount the stamina of offspring and favor the early limitation of endurance and vigor demonstrated in neurasthenia. A defective *education* that omits discipline and the cultivation of self-control, poorly fitting the child for the rude shocks of later life, may be a predisposing cause. Educational methods that overtrain and overstrain may directly induce the neurosis. *Occupations* of all varieties furnish neurasthenics. It is only requisite that the element of overwork come in, whether the labor be mental or physical. Overwork, moreover, is a variable quantity, relative to the forces and endowments of the individual.

Among the *inciting causes overwork* is the most common, and associated with it we usually encounter *anxiety, worry,* or *excitement,* which depress the patient's forces, often at the same time impelling him to greater efforts. The business man, anxious for his ventures, works doubly hard to secure success. The sleepless mother, worn with care and nursing, does double and treble duty, and finally "goes to pieces" when the strain is over. The overtrained athlete goes "stale." *Excesses,* by their debilitating effects, are frequent sources of neurasthenia. Alcohol, tobacco, venery, masturbation, either as onanism or withdrawal, unnatural stimulation of sexual responses, and, very rarely, sexual continence may result in the general depression we call neurasthenia. *Trauma,* both physical and mental, may induce neurasthenia, and is likely to do so in proportion as the psychic shock is well developed. Railway accidents may breed neurasthenia in those who do not receive a scratch. The fright is often worse than the blow. Together they may cause a double injury. A muscular strain from overexertion, as in lifting, may start neurasthenia, particularly if the back be hurt and there is suggestion or fear of serious harm having been done. The more neurasthenia is studied, the more prominent will its mental side become. Exhausting *illness,* either from acute or chronic disease, may cause neurasthenia. Various *toxic states,* such as lithemia and syphilis, are prone to produce it. It is usually difficult, and often impossible, to determine the exact cause of neurasthenia in a given case. Ordinarily, there are a number of both predisposing and exciting causes. In a large number of instances neurasthenia is *secondary* to and *symptomatic* of organic conditions, such as phthisis, Bright's disease, diabetes, gout, rheumatism, uremic and toxic states generally.

Pathology.—Although we know no pathological anatomy of the disorder, its manifestations are those best explained by a diminished dynamic energy and lessened recuperative power in the cerebrospinal axis, and especially in its cellular elements. This may be a nutritional defect. The very constant factor of persistent overwork, overstrain, and overfatigue in the causation of neurasthenia, coupled with Hodge's findings in the motor cells after an expenditure of energy, leads to the opinion that we have to do with a fatigue neurosis, general in distribution and comparatively slight in degree. With this view in mind, we will be the better able to understand the symptomatology and the requirements for treatment.

Symptoms.—The symptoms of neurasthenia are extremely numerous. Some of these are *essential,* most are *adventitious.* Charcot considered *headache, backache, gastro-intestinal atony, neuromuscular weakness, cerebral depression, mental irritability,* and *insomnia* as the fundamental symptoms of the disorder,—the true stigmata of the neurosis. Secondarily and inconstantly arise a host of complaints that are of less importance and significance. It will be necessary to take up the symptoms seriatim. It may be said of them all that they indicate a deficiency of function, never an absolute want of it. The reduction is one of quantity rather than of quality, but is never absolute in degree.

Motor Disorders.—A constant condition in neurasthenia is *muscular weakness.* The patients complain that muscular efforts are promptly fatiguing. They can only walk a few squares, standing a long time is exhausting, and exercise or use of the back and upper extremities prostrates them. They often show a remarkable diminution of strength as registered on the hand-dynamometer or in lifting, but occasionally a patient can put forth one or two fairly forcible efforts, and then the strength quickly subsides. Sustained effort is impossible. As another manifestation of the generalized myasthenia, *tremor* is frequently observed, and can usually be provoked by comparatively slight muscular efforts. Lamarcq[1] found it in eighty-five per cent. of neurasthenics. Many patients complain of their trembling knees and shaking hands, or the tremor may appear in the handwriting, especially toward the end of a long letter, where the firmness of the strokes is also likely to be reduced. Tremor in the lips and face is sometimes noticed. Muscular *twitchings* in the face and extremities are not rare. The *tendon reflexes,* ordinarily, are increased. This is customarily the case with the knee-jerk. A tendency to widely distributed responses is frequently present, so that a tap on the patellar tendon causes starting of both lower limbs or of all four extremities, and sometimes gives rise to a complaint of pain in the back. If the knee-jerk be repeatedly produced, the intensified response first elicited may gradually subside and even disappear, giving another evidence of the early fatigue of the nervous apparatus. In some instances a tap on muscle or nerve-trunk will call forth a similar response. Ankle-clonus is sometimes present, but is spurious in character, and only a few vibrations of the foot can be elicited. Repetitions of the test may fail to produce it. Paralysis or abolished knee-jerks are not found in neurasthenia unless due to other coincidental disease.

Sensory Disturbances.—Neurasthenia never causes *anesthesia.* When this is present we have a condition of actual deficit, to which limit neurasthenia does not go. The vague subjective disturbances of sensation, on the other hand, are limitless. Among them a feeling of *general tiredness* and *fatigue* is almost constant, and the recumbent posture tends to become habitual.

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[1] "Revue Neurol.," August 15, 1896.

Headache is one of the most common symptoms; it is practically never wanting and often is described in striking terms. In some instances it is slight and constant, more often it is produced by any muscular or mental effort, and occasioned by any disturbing emotion. Usually it is occipital, "at the base of the brain," as these patients are fond of saying; but it may be frontal, temporal, or vertical. One describes a sensation as if the head were splitting or the skull lifting; another has a terrible weight or a severe constriction about the head. This *lead-cap headache* is very common. Heaviness, throbbing, buzzing, a sensation of wind blowing or of water running under the scalp, and many others are perhaps indicative of the vasomotor disturbances within the skull. *Backache* is equally common with headache. It is probably a fatigue symptom for the most part, though sometimes referable to gastro-intestinal disturbances. The small of the back is its usual seat, whence it streaks up between the shoulders or through the loins and down the limbs. Very commonly it is associated with a "drawing" sensation at the back of the neck, and the occipital pain is then usually complained of. By lying down or by padding a chair with pillows, some relief is obtained. The advanced case customarily assumes one of these expedients. In milder cases the backache is occasioned by any effort or disturbing circumstances and subsides upon rest.

Tenderness is usually found over the spine and is rather superficial in character, but occasionally is intensified by deep pressure. Rarely the spine is tender its entire length; usually only small sensitive spots are present. The upper cervical spine near the occiput, over the vertebra prominens, opposite the lower angle of the scapula, at the waist-line, at the top of the sacrum, and over the coccyx are the favorite locations. Sometimes the tenderness is diffuse and the skin over the entire dorsum is painfully over-sensitive. Sometimes this sensitiveness prevents the dorsal decubitus or interferes with sitting up. It is likely to be aggravated by anything which disturbs the patient or increases the other symptoms. Sensitiveness on the head, limbs, or other portions of the trunk is not rare, and is usually associated with *spontaneous pain* in the same location. The outlines of these sensitive areas are never sharply defined, and they may shift position or vary in intensity within a few hours or days, but sometimes remain practically stationary for months.

All manner of vague sensations of heat, cold, prickling, tightness, numbness, stiffness, weakness, fatigue, soreness, pain, pressure, etc., referred to this or that part of the body or limbs are constantly encountered. In addition there are a host of abnormal feelings referred to the thoracic, abdominal, pelvic, and generative organs.

Visual Disturbances.—The neurasthenic commonly complains that reading has grown difficult because it causes headache and vague distress, and states that the letters blur or run together after a few minutes. In other instances they can not maintain attention. Careful examination will usually demonstrate that the *accommodative apparatus* and the *retinal sensitiveness are promptly fatigued.* One is a motor loss, the other a sensory deficiency. The first is shown by the rapidly diminishing ability to clearly make out the test-type; the second, by the quickly narrowing visual field, due to impaired sensitiveness in the retinal periphery. Both may be regained after a slight rest. A high and oscillating degree of muscular asthenopia is usually due to neurasthenia, and commonly subsides as the nervous state improves. Deficiency of the interni is the usual finding.

Photophobia may be encountered of sufficient intensity to keep patients in dark rooms or wearing colored glasses. In lessened degree retinal hyperesthesia is not uncommon, and is similar to the cutaneous sensitiveness. Misty obscurities and a veiling of vision are sometimes described, or everything looks strange and unreal. The *pupils* are usually very mobile, contracting and expanding excessively, sometimes sluggishly, sometimes very actively and even independently of light or accommodative efforts. Inequalities, both transient and persistent for days, are rarely encountered. Permanent inequality is due to organic disease.

Disorders of Hearing, Smell, and Taste.—Hearing is apparently frequently disordered. Thus, patients start at the slightest sound, and often tie up the door-bell and seek seclusion to avoid all such irritation. This is as much mental as aural, perhaps, but tinnitus in various forms and throbbings in the ears are due to the irritable weakness that spares no nerve. Complaints of peculiar or bad smells and tastes have a similar significance.

Gastro-intestinal Disorders.—*Nervous indigestion* is one of the commonest features of neurasthenia. The appetite is often capricious, and may be excessive or greatly diminished. The mere thought of food may be repugnant. In milder cases there are complaints of the food lying heavy or of gaseous eructations, which may or may not be attended by heart-burn. The gaseous gastric distention may suggest dilatation, and often provokes cardiac palpitation and precordial and epigastric distress. In more aggravated cases dilatation actually occurs, and hydrochloric acid may disappear from the gastric secretion. Digestion is retarded and deficient, but the tongue may remain clean and nutrition still be inadequate to maintain the body-weight. The *small intestine* is usually affected in the same atonic fashion, causing constipation. In the severe cases of neurasthenia all these conditions are aggravated. The eructations of gas become frequent, noisy, and distressing; meteorismus, colicky pains, alternating constipation and mucous diarrhea, intestinal fermentation, and the passage of undigested food mark the atonic and irritable state of the gastro-intestinal tract. Cases present great variations, and the same case is rarely consistent in regard to these features.

Circulatory Disorders.—*Cardiac palpitation* due to digestive disturbance is one of the common symptoms in neurasthenia, but may result from other causes, as muscular or mental efforts, sudden starts or embarrassments of any sort. In some instances it reaches a high degree and is attended by precordial pain, a tumultuous heart's action, throbbing arteries, and generalized distress. In other cases it may occasion a pseudo-angina pectoris that mimics a stenocardial attack very closely. A small, rapid *pulse* of about 90 a minute is commonly present in neurasthenia, and in attacks of palpitation it may reach 140 or 160. The rate is promptly increased in all cases by any distressing, exacting, or disturbing cause. Hemic murmurs are not rare in advanced cases, when the general nutrition has been reduced. The *feebleness of the circulation* is shown by the cold extremities commonly encountered. *Vasomotor storms* in neurasthenia are the rule. Localized or general flushings, sweats, and aortic throbbing may greatly annoy the patients.

Secretory Disorders.—The *urine* is ordinarily scant and high-colored. Neurasthenics drink little water, and the irritating urine may cause much vesical uneasiness and frequent micturition. There is commonly an increase of uric acid and urates, or a great abundance of phosphates and oxalates may be noted, and the urine may be neutral or alkaline in reaction. All *fluid secretions*, as the perspiration, saliva, gastric, intestinal, and synovial fluids may be deficient. In other instances they are increased, or increase and deficiency may alternate. Some patients complain that the slightest effort or embarrassment causes them to sweat profusely, usually about the head and neck. The cold, clammy hand of neurasthenia is rarely wanting.

Genital Disorders.—The average male neurasthenic complains of *lessened sexual power*. If unmarried, nocturnal emissions frighten him, and he is convinced that he has spermatorrhea by the cloudiness of his urine. If, perchance, there has been an antecedent habit of masturbation, every symptom is warped by him into relation with the supposed impotence, and he develops into a confirmed sexual neurasthenic without erections and with vague paresthetic sensations or actual tenderness and pains in the genitals that claim his undivided, brooding attention. If married, premature ejaculations and loss of sexual appetite in milder cases are followed by actual inability in the severer forms, and some sensory hyperesthesia or lessened sensitiveness of the parts may be found.

In women analogous symptoms are encountered, but much less frequently. Some are troubled by nocturnal orgasms accompanying dreams from which they awake nervous, depressed, and exhausted. In married women sexual appetite may at first be somewhat increased, but quickly diminishes and commonly disappears, not seldom being followed by an actual distaste or even disgust. Pelvic pains and genital insensitiveness, or hyperesthesia, are often added, and further serve to inhibit intercourse.

Mental Disturbances.—The psychic side of neurasthenia is an interesting and important feature of the neurosis. All the mental manifestations of the neurasthenic have in common the same elements which dominate the physical features—namely, weakness and irritability. The *capacity for mental work is abridged*, just as the muscular power is reduced. Protracted mental efforts become irksome or impossible, and in many instances attempts to read or think or converse for a few minutes produce so much fatigue and discomfort that they must be discontinued. This *mental asthenia* also shows itself in the reduction of the mental concentration necessary for fixing and maintaining attention.

Neurasthenics can not keep their minds on the printed page nor follow a line of thought for any great length of time. They are inattentive to details, and consequently do not clearly apperceive them. Hence arises the almost constant complaint of a *loss of memory*. Active *spontaneous mentation*, which is dependent upon mental strength, is also reduced. Ideas do not occur to such patients with their usual vigor and rapidity, and they often assert that they can not think. Their *courage* naturally subsides with their strength. Attracted by the cardiac palpitation or their sexual incapacity or their mental debility, or by any other neurasthenic index, they become *introspective*, and, misconstruing their symptoms, develop a lot of *nosophobias*. As the suggestions and reminders of these fears are constantly present, the neurasthenic becomes saturated with them, thus still further reducing the power of attention and memory. They become morbidly self-watchful and tend to hypochondriacal *depression*. In some instances these morbid fears pass into insane delusions and obsessions, and are then beyond the boundaries of neurasthenia, but the dividing line is decidedly indefinite. Indeed, many psychoses begin as a neurasthenia and even general paresis may show a neurasthenic prodromal period.

Fear, arising easily from a consciousness of weakness and a loss of courage, often becomes associated with external conditions and suggestions. An attack of palpitation in a crowded place may induce a condition of fear or apprehension constantly associated with like conditions. Some fear to be alone, others to be in open or in narrow or in high places. Some fear darkness, some storms, some lightning, some special localities, some contamination by dirt or infection by disease. The neurasthenic always recognizes the baselessness of such fears, though he may not be able to dispel them; the insane hypochondriac accepts them as actual necessary facts, and can not be persuaded to the contrary.

Frequently the neurasthenic recognizes a *diminished affection* for his family or others, and sometimes is greatly troubled by it. He is irritable, peevish, fault-finding, and resentful. The business man dreads his daily tasks and dislikes to meet new customers or even old friends. The *emotions* are less stable, and there is ever a tendency to weakness and depression. Such patients are lacrymose on slight occasion or break down in telling of themselves or in contemplating their darkened future.

Sleep is commonly faulty from the first. Neurasthenics habitually present *insomnia*. In some instances there is a difficulty in getting to sleep or the sleep is constantly disturbed. Exceptionally the sleep is sound and prolonged, but the patient *awakes unrefreshed* and usually more depressed and prostrate than upon retiring. Ordinarily, the sleep is disturbed by troubled *dreams*, which often are of a dreadful or nightmarish character. In shock or traumatic cases the accident episode may be repeated in the dreams night after night or several times the same night. Charcot laid especial stress upon such formulated dreaming in neurasthenia.

The General State.—In neurasthenia of long standing the general nutrition is commonly defective, and high degrees of anemia and emaciation may be encountered. Neurasthenia of a moderate grade of severity, but sufficient to disable the patient for ordinary pursuits, is consistent with fair nutrition, and some of the most inveterate cases may become quite fat. Emaciation, on the one hand, is not a hopeless indication, nor, on the other hand, does plumpness indicate an easily manageable case.

Forms.—In view of the multiplicity of symptoms in neurasthenia and their lack of uniformity in various patients, it is not surprising that numerous forms have been described. The terms *cerebral, spinal,* and *sexual* neurasthenia have been used to designate cases in which symptoms referable respectively to the head, back, and genitals have preponderated, but it is best to look upon neurasthenia as one and indivisible. Adventitious or exaggerated symptoms may variously tone the clinical picture in different cases, but neurasthenia remains a generalized disorder of the entire nerve-apparatus. In the foregoing description of neurasthenia the simple variety has been constantly in view. We are always to have in mind that neurasthenia may be *symptomatic* of some organic process of which it is a secondary expression. Thus, it may follow all forms of extraordinary illness or injury, spring from the various cachexias, and succeed all toxic and infectious processes. All such conditions must be eliminated before we may call a case one of *simple* or *essential* neurasthenia. Again, neurasthenia is frequently encountered in *association* with other neuroses. This is particularly true of hysteria, and it is sometimes impossible to say where the neurasthenia ceases and the hysteria begins. For such cases the term *hysteroneurasthenia* has been employed.

Course.—Neurasthenia is essentially a *chronic malady.* Ordinarily, its *onset* is insidious, and it is only after weeks and months of growing incompetency and laborious efforts to carry on the usual duties that the patient recognizes something wrong and seeks medical advice. In the traumatic form of neurasthenia the neurosis may be quickly established, but it is not uncommon for the patient to go about as usual for several days or weeks after the accident or injury, and gradually develop the nervousness, weakness, and prostration. Once established, neurasthenia tends to persist indefinitely, and only some radical change in the mode of life, serving to relieve the patient of worry and work, is likely to favorably modify the condition. Frequently remissions are presented, but the patient relapses under any unusual demands, and slowly or partially regains the former status. Even after long periods of improvement there is a tendency to ready recurrence under the influence of any exciting cause.

Diagnosis.—The diagnosis of neurasthenia is usually easy. The combination of mental and physical weakness and irritability with gastro-intestinal atony, backache, headache, insomnia, and rapidly exhausted retinal sensitiveness, justifies the diagnosis. The difficulty mainly lies in overlooking some basic organic disease. One should never be content with a diagnosis of neurasthenia until he can thoroughly satisfy himself that he is not confronting a symptomatic form of the neurosis. *Hysteria* may be associated with neurasthenia, but has its own stigmata.

It is to be kept in mind that disturbances of function in neurasthenia are those of irritable weakness and not of actual loss. Anesthesias, palsies, convulsions, complete loss of self-control, actual amnesias, are not the property of neurasthenia, but are the ear-marks of hysteria. True *hypochondriasis* is a variety of melancholia with actual insane delusions regarding bodily states. In neurasthenia the phobias are under at least partial control, and sometimes can be completely relieved by a reasonable statement of the facts. As before stated, all the changes of function in neurasthenia are in the nature of quantitative reduction.

Prognosis.—The outlook in neurasthenia is commonly good if the proper treatment can be instituted and maintained. It is often very difficult to secure the coöperation of the patient, or his circumstances may be such as to preclude the protracted rest and change usually required to restore him. In that event he worries along as best he can, possibly picking up some drug or stimulant habit, and may recover after many months, but is more likely to break down completely and become a confirmed nervous invalid. If of a psychopathic stock, some pronounced mental change may appear. Cases occurring before twenty years of age are likely to yield readily and to relapse frequently. Such early demonstration of insufficient stamina naturally indicates an unstable organization. After forty the prognosis is also less hopeful, as the neurasthenic exhaustion occurs in tissues that have largely lost their resiliency and recuperative powers. After this age full vigor is practically never regained if the neurasthenic state has been severe. A neuropathic or psychopathic heredity or evidence of degeneracy in the patient reduces the chances for a full and permanent recovery. The bodily condition is of less significance if organic disease is absent. Cases of extreme emaciation often respond admirably to treatment, while some of the cases without marked disturbance of bodily function are inveterate.

Treatment.—The keynote of treatment in neurasthenia is *rest.* Depending upon the severity of the case, upon the temperamental characteristics of the patient, and upon the sex, this rest may be partial or as complete as we can possibly make it. In the milder cases, especially those occurring from overwork in sedentary pursuits, in patients between the ages of twenty and forty, and when the patient's make-up is based upon a good heredity, the full Wier Mitchell rest treatment is commonly not required. It may only be necessary to have the patients spend four or six additional hours in bed daily. This can be secured by having them retire shortly after the evening meal, and not rise until late in the morning. A hot bath of five minutes' duration and a glass of hot milk or a glass of beer on retiring for the night encourage sleep. A cold douche or cold sponge, to the full length of the spine on rising, gives a vigorous morning start. In addition, an hour's rest in the recumbent posture in the middle of the day should be enjoined. The amount of *work must be reduced.* It is rare that this can not be done even in the most responsible positions. It should be needless to say that the condition of *organic activity,* if deranged, must be put to rights. Constipation, a sluggish skin, or inactive kidneys, if uncontrolled, will defeat the best general management. Large quantities of drinking *water* usually aid in several ways, and, as a rule, neurasthenics shun water. If these regulations can be instituted during a *vacation*, especially one spent among *new scenes*, an additional advantage is gained.

In the *severer male cases* an absolute *separation from business* and family is usually required, and a long *sea voyage* with a pleasant companion often works wonders. As a rule, the more *outdoor air* and *recreation* that does not entail effort, the better, but to put a neurasthenic on a bicycle or on long walks adds fuel to the flames. A carefully selected, *nourishing, fattening, unstimulating diet* is of considerable importance. If the scales show a gain of a few pounds, success is assured. Men, unless practically bedridden, do not respond favorably to the Mitchell rest-treatment. The enforced idleness and confinement to bed is rarely tolerated by them if they can possibly be about.

In the *severe female cases*, and sometimes in young males or completely prostrated men, we must have recourse to the full rest plan laid down by Mitchell. The details of this are furnished in all modern works on therapeutics and need not be repeated here. By this plan *expenditure of energy is reduced* to a minimum, *strength is conserved and increased* by the massage and electricity, and *flesh is rapidly made* by the constant feeding. There are several essentials in carrying out this procedure, and their neglect means failure. The first is *isolation* from relatives and friends, and the establishment of an entirely new and *hopeful atmosphere*. In some instances the slightest infraction of this rule causes immediate relapse. Equally important is it to have an experienced and tactful *nurse*.

Any amount of general hospital training does not make a good nurse for this class of patients or furnish the endless tact and self-repression needed to manage them. It is difficult to induce nurses, accustomed to grave operative cases and severe physical illness, to appreciate that the endless complaints and fault-findings of neurasthenic patients are symptomatic of their state and not merely petulant crankiness. It is also very important that the treatment take place in a *suitable location*, free from disturbing noises. The room and immediate surroundings of the patient must be bright and cheerful. If isolation with a suitable nurse, preferably one able to give massage, can be had, the other details of treatment are less important. The absolute milk *diet* does well in some cases, and is indispensable in a few at the beginning, but if food is digested and the bowels active it is immaterial what the patient eats so long as it is nourishing, abundant, and administered at frequent intervals. Sleep, usually disturbed, under the massage and forced feeding soon becomes sound and prolonged. Hot milk, an alcohol rub, or a glass of beer at bedtime is commonly a sufficient hypnotic.

No scheme of treatment in neurasthenia is complete that ignores the *mental element* of the disorder. These patients are frequently impressionable, and particularly so as regards their own health and prospects. Their fears can not be laughed to scorn. They should be carefully estimated, fully explained, and then dismissed. It is not advisable to allow these patients to reiterate their complaints to the physician and the nurse, and the family must be instructed to refuse to hear or discuss them with the patient. *Encouraging* suggestions and *hopeful* assurances *constantly repeated* have the force of hypnotic suggestion, and a very real therapeutic value. For this reason the physician must usually see his patient frequently. In this way the daily use of static electricity, hypodermic injections of strychnin or water, or any other objective excuse, impresses the patient and permits and emphasizes the repeated encouraging suggestions.

Thus far nothing has been said about *drugs*, and there is very little to say. Iron and arsenic against the anemia, trional and bromid for the insomnia, laxatives for constipation, may be given. Strychnin in large doses, or bromids and sedatives in large doses, are ill-advised and commonly harmful. The indications are to rest and to nourish, not to stimulate or to depress. *Hydrotherapeutics*, especially if carried on at a distance from home, yield good results in mild cases. It may be said that the treatment of neurasthenia requires judgment, tact, perseverance, and personal force of the highest order. When the neurosis is secondary to organic processes, its treatment is similarly second in order. *Climatic conditions* embracing windy low-lying stretches, altitudes above 3000 feet, and fluctuating extremes of temperature are decidedly disadvantageous. As a rule, neurasthenics do best at the sea-level and preferably at the seaside.

CHAPTER VI.

HYSTERIA.

HYSTERIA has been an interesting problem since the earliest days of medical thought. Unrecognized it occasioned the demoniacal "possessions" of the middle ages, and furnished some of the martyrs of witchcraft and religious fanaticism. Affecting whole communities, it caused epidemics, allayed by appeals to St. Guy, St. Vitus, or other tutelary. It has been seen in the excited religious gatherings of all countries. As a disease it was long supposed to be limited to the female sex, and was attributed to vapors or other influences arising from the womb; hence the name hysteria. Only in recent years have the endemic and epidemic forms been understood, and the male found to share with the female in the liability to the psychoneurosis. For many years the mental element in hysteria has been at least partially recognized. Moebius used the definition, "A state in which ideas control the body and produce morbid changes in its functions." So far as the paralyses and contractures are concerned, English writers have described them as "depending upon idea." The studies of Charcot and his students have placed hysteria upon a firm clinical basis, and enabled nearly all of its manifestations to be traced to disturbances in the psychic sphere or in its substrata. Janet says: "Hysteria belongs to a group of mental diseases of cerebral insufficiency; it is especially characterized by moral symptoms; the principal one is a weakening of the faculty of psychological synthesis." "It results," adds Dutil, "that a certain number of elementary phenomena, sensations, and images are not preserved, and appear to be repressed in the realm of consciousness." In addition, there are a number of organic phenomena—disturbances of nutrition, trophic and vasomotor disorders—of a neurotic character. Hence, we class hysteria as a psychoneurosis. The absence of detectable primary changes in the nervous system is admitted on all sides even in very chronic cases.

Etiology.—Hysteria in slight or severe form is one of the most common of nervous diseases. The *age* of puberty and the years of adolescence immediately following furnish the majority of cases. After twenty-five the frequency of hysteria declines and it becomes rare after forty-five. Before ten it is also uncommon, but children may develop it in very marked form even as early as two or three years of age. Formerly considered almost exclusively limited to the female *sex*, later statistics go to show that males and females are affected with hysteria in nearly equal ratio. According to Marie, in the lower social levels males predominate; in the wealthier classes females are more commonly affected. Hysteria is a disease of all *countries* and all *races*, but the Latin, Slav, and Israelite may be considered as particularly liable.

Heredity plays an important part. Hysterics usually belong to neuropathic families. Hysteria in the mother is very frequently followed by hysteria in the daughter. More commonly, however, the transmission is by *transformation* from, or to, other neuroses and psychoses. *Arthritism* or *phthisis* in the antecedents of hysterics plays the same part as in other manifestations of degeneracy.

Inciting Causes.—*Emotional disturbance* of any sort may initiate hysteria. Fright, grief, worry, chagrin, and every sort of mental and moral strain and shock are the common starting-points of this multiform disease. *Traumatism* furnishes a large quota of hysterics, especially of the male sex, owing to their greater liability to such accidents. In all such circumstances, unless consciousness be abolished instantly without preceding anxiety or fright, the attending psychic states must be taken into consideration. As a practical fact, the likelihood of hysteria following trauma is in direct proportion to the intensity of the mental shock. The physical injury may be insignificant. Lightning-stroke, surgical wounds, and internal conditions, such as gastric ulcers, nephritic and hepatic colics, may act as causes.

Intoxication by lead, mercury, sulphid of carbon, oxid of carbon, tobacco, morphin, cocain, and chronic alcoholism, or even a single alcoholic debauch, may induce hysteria. In many such cases these intoxications furnish a basis on which hysteria develops by the incidental action of some other provocation. *Infectious diseases*, such as typhoid, diphtheria, influenza, pneumonia, scarlatina, malaria, and syphilis, may provoke hysteria. It may occur in *cachectic states* due to chlorosis, diabetes, phthisis, and cancer. It is found as an *associate* of all organic diseases of the brain and spinal cord, frequently appearing in tabes, syringomyelia, and insular sclerosis. Either mental or physical *overwork* may cause it. Wherever people of suitable age are domiciled together, hysteria may become endemic through the force of *imitation* and suggestion arising from an initial case of hysteria or of some physical disease. Schools, prisons, barracks, and large families may thus become affected. Usually in such instances there is great similarity among the cases. In this country, under the prolonged excitement and fervor of protracted religious meetings in rural districts, endemics of hysterical spasms and even of dancing, in all respects similar to the medieval epidemic dance of St. Vitus, have developed. Hysterical patients in hospitals may closely *mimic* all the symptoms and physical disabilities of other patients with whom they are kept in contact. In other cases they reproduce the manifestations of some disease with which, in their past experience, they have been made familiar.

Symptoms.—The innumerable symptoms of hysteria, to follow the plan of the French writers, may be divided into two major groups: those which are essentially persistent,—the *stigmata;* and those which occur incidentally, are intermittent or transitory,—the *accidents* of hysteria. The stigmata are not necessarily present singly or in combination, but, once developed, tend to persist so long as the affection lasts. The accidents present the greatest diversity in different patients, but usually, if they occur repeatedly, tend to uniformity in a given case.

Further, some hysterical accidents, as paralyses or contractures, may be of long duration, and, once thoroughly established, have the force of stigmata.

STIGMATA OF HYSTERIA.

The stigmata of hysteria are *sensory*, *motor*, and *psychic*.

Sensory Stigmata.—In hysteria the sensory disorders are (1) of the negative variety,—anesthesias; and (2) of the positive sort,—hyperesthesias. They are usually both represented in a given case, but the anesthesias are the more important symptomatically.

Hysterical anesthesia may affect sensation in all its modes and tenses, including the special senses. The general cutaneous sensibility may be disaggregated so that only certain elements persist and a limited group of stimuli alone serve to arouse the sensorium, as in a thermo-analgesia that parallels the sensory dissociation of syringomyelia. The diminution of sensibility may be partial or complete, and often varies in the same patient within a very short time. Various degrees of anesthesia may likewise be found in different regions, and the anesthetic area represents remarkable variations of extent and distribution in different cases, and also in the same case at different times. Some form and degree of anesthesia is rarely lacking in hysteria that has existed any length of time, and often it is developed very early. It is obligatory to persistently search for it in every instance, but care must be exercised not to induce it by suggestion. As a rule, hysterics are themselves ignorant of their sensory deficiencies. The anesthesia may be (1) superficial, affecting mainly the skin and mucous tissues, or (2) it may involve the deeper structures.

Cutaneous anesthesia may be absolute. Pricking, pinching, hot and cold bodies, produce no response. Some patients are merely analgesic, and this is the common defect. Less frequently there may be thermo-anesthesia or thermo-analgesia, and, most rarely of all, tactile sensations alone may be wanting. Very exceptionally the hysterical patient feels the faradic current in the anesthetic area or, more rarely still, presents an area insensitive to this stimulus alone. Complete anesthesia, hypesthesia, and analgesia are the commonly encountered forms. The *mucous membrane* within the range of examination may show the same anesthetic modifications of sensibility. The buccal, pharyngeal, laryngeal, nasal, conjunctival, anal, urethral, and vaginal surfaces may be entirely insensitive or present dissociation of sensation.

The *deeper parts* are frequently anesthetic. Bones, muscles, ligaments, and nerve-trunks may sometimes be pierced, twisted, wrenched, and contused without giving rise to distress or even provoking a localized sensation of any sort. The *muscular sense* for a limb may be abolished so that with bandaged eyes the patient has no knowledge of its position, can not estimate weights, recognize pressure, or feel fatigue.

The Special Senses.—*Taste* and *smell* may be perverted, diminished, or abolished. Certain sapid articles may fail to arouse the sense of taste, while others are still detected. The loss of taste is usually limited to a portion of the tongue and mouth. *Hearing* is often greatly diminished, but complete hysterical deafness is very uncommon. Rinne's test (see p. 64) shows the disturbance to be central. When the loss of hearing is pronounced, one commonly finds more or less anesthesia in the auditory canal and on the same side of the head and face, but absolute anesthesia of the drumhead is exceptional.[1]

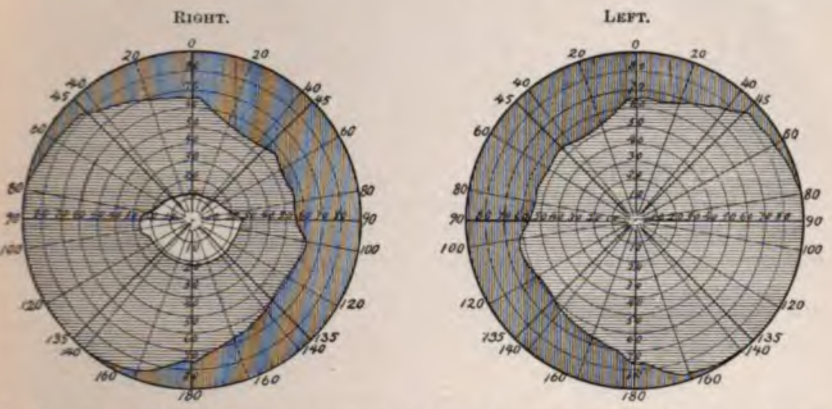

Fig. 246.—Hysterical concentric contraction of visual field of right eye; amaurosis of left eye (Tourette).

In hysteria *vision* is very frequently modified, and some of the changes in this special sense are of the utmost importance for diagnosis. Complete blindness is very rare, usually of abrupt onset, a few days' duration, and sudden recovery, but vision is often reduced in one eye to counting fingers or less. Of greater frequency and of more importance are the lesser and commonly persistent defects. These consist of: (1) A reduction of the field; (2) troubles of color perception, and (3) errors of accommodation.

Contraction of the Visual Field.—In the great majority of hysterics the visual field is *concentrically* contracted. This is usually found bilaterally, but commonly more on one side than the other, and sometimes only on one side. The defect, as in that of all the special senses, when unilateral or most marked on one side, usually corresponds to a unilateral distribution of disturbed cutaneous sensibility, but the opposite situation may be encountered. In total amblyopia the contraction of the field reduces it to zero. In a given case the retraction of the field is practically permanent, but it may fluctuate greatly. An epileptiform attack, fatigue of the eye, emotions, and variations of attention may modify its limits. Hysterical hemiopia and scotomata are so infrequent that they should always suggest an organic lesion.

The *dyschromatopsia* is more characteristic of hysteria than the contracted field, which is also present in neurasthenia. In the normal eye the fields for the various elementary colors are not coextensive (see p. 63). In their natural order blue has the largest field, followed by yellow, orange, red, green, and violet. In hysteria not only are the fields for form and light perception contracted, but those for colors are also diminished, and to a proportionate degree. The important particularity is that the normal order is generally changed. The *red field often exceeds the blue.* When the color-fields are extinguished, they usually disappear in this order: violet, green, blue, yellow, and, *last of all, red.* Objects are then finally seen as grayish, and we have the condition of *achromatopsia.*

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[1] Chavanne, Paris, 1901, "Oreille et Hysterie."

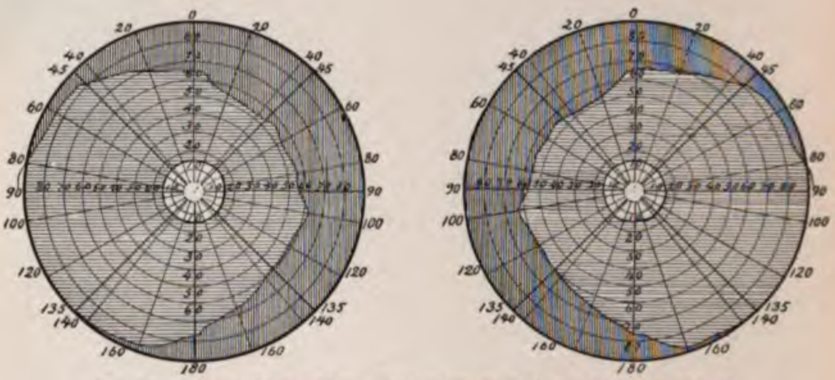

Fig. 247.—Hysterical bilateral concentric contraction of visual fields (Tourette).

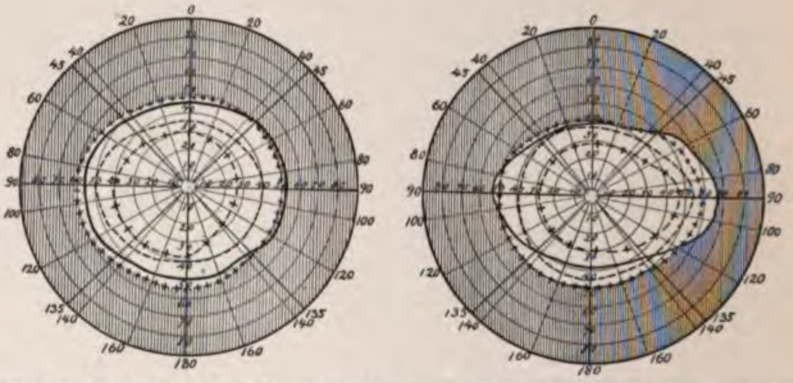

Fig. 248.—Concentric retraction of visual fields for colors usually found in hysteria. Red field inclosed thus: + + +; white field, ——; blue field, — — — —; green field, + — + — + (Souques).

Accommodative errors in hysteria are sometimes encountered, by which near vision, particularly, is rendered faulty. To the same source have been attributed the *monocular diplopia, polyopia, macropsia,* and *micropsia* that are occasionally met. In this condition, with the opposite eye covered, the patient fixes the gaze upon a pencil close to the eye. As it is withdrawn, its image doubles, and at a greater distance a third may appear, and they all increase in size, and again diminish as the pencil approaches. Errors of curvature in the cornea must be excluded to render the symptom significant.

It has recently been recognized that, aside from complete bilateral blindness, the various forms of hysterical amblyopia are not present in *binocular vision.* Even opening the unaffected eye, in the case of unilateral amblyopia, at once widens the contracted field (Parinaud). Patients who can not read with either eye alone may do so readily with both. That the amblyopic eye sees is easily demonstrated. A red glass over the affected eye and a prism over the other double the image of a flame, and the red image, corresponding to the amaurotic eye, is distinctly seen by hysterics. Or, in a case of unilateral green-blindness, a prism doubles the image of a green object and both are perceived as green. Again, an eye that fails to detect everything but red, upon looking at a revolving Newton wheel bearing red and green, sees it to be whitish, as does the normal eye, proper cognizance having evidently been taken of the complementary green. It is evident, therefore, that the trouble is not in the eye or in the paths of conduction, but that, under ordinary circumstances, the impression fails to enter the field of personal consciousness. These symptoms are unattended by any change in the retina or media. Spiller[1] insists that the *lachrymal reflex* remains in hysteria even when the eye-parts and the side of the face are anesthetic, while in organic lesions the tears are not similarly secreted upon irritating the conjunctiva, as with a piece of paper placed under the lid.

Distribution of Hysterical Anesthesias.—In a very small proportion of cases the anesthesia in hysteria involves the entire cutaneous and mucous extent. Usually it is limited (1) to one-half of the body; (2) to areas of more or less definite geometric outline; and (3) to discrete islets.

Hysterical hemianesthesia is a common distribution of sensory deficiency. Ordinarily, it affects the left half of the body, and is sharply limited by the median line. If intense, the approachable mucous surfaces on the same side are commonly also anesthetic. As a rule, the special senses—sight, hearing, smell, and taste—are blunted on the anesthetic side, but in some cases the special senses are affected on the opposite side.

Anesthesia in geometrical areas is frequently encountered. An entire extremity or a band about an extremity may alone be anesthetic. The distribution may often be described as a sleeve, glove, sock, stocking, or drawers-leg anesthesia. The outlines have no relation to the anatomical distribution of the nerve-trunks whatever, but are referable to functional groupings. They often have a superimposed relation to functional disturbance, as in cases of paralyzed or contracted limbs, diseased joints, or local injury. In hysterical amblyopia the conjunctiva, lids, and a circular area about the eye are usually anesthetic. In hysterical deafness the auditory canal and concha are frequently insensitive. In hysterical aphonia the larynx may be anesthetic.

Islets of anesthesia of peculiar and paradoxical outline are sometimes encountered. These may be many or few, and it often requires minute and painstaking search to find them.

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[1] "Phila. Med. Jour.," May 17, 1902.

Peculiarities of Hysterical Anesthesia.—1. In the first place, the outlines of limited hysterical anesthesias, excepting hemianesthesia, do not conform to the anatomical distribution of cutaneous nerves or to the sensory areas related to the vertebral segments, but rather to the mental association of functions, and probably to their cortical representations.

2. The *organic* and *tendon reflexes* in hysterical anesthesia are not modified, as in organic lesions marked by insensitiveness. The *pupil* responds to light and accommodation and to pinching of the neck, even when the eye is amblyopic and the skin of the neck is insensitive. The *abdominal, cremasteric, knee,* and *toe* reflexes are not abnormal. The *cardiac* and *respiratory* reflexes to painful cutaneous stimulation are retained, even when an anesthetic zone is used. Sometimes a spurious ankle-clonus can be obtained. Other reflexes dependent principally upon sensation are abolished by hysterical anesthesia. The *palpebral* and *pharyngeal* reflexes disappear if the parts are insensitive. The

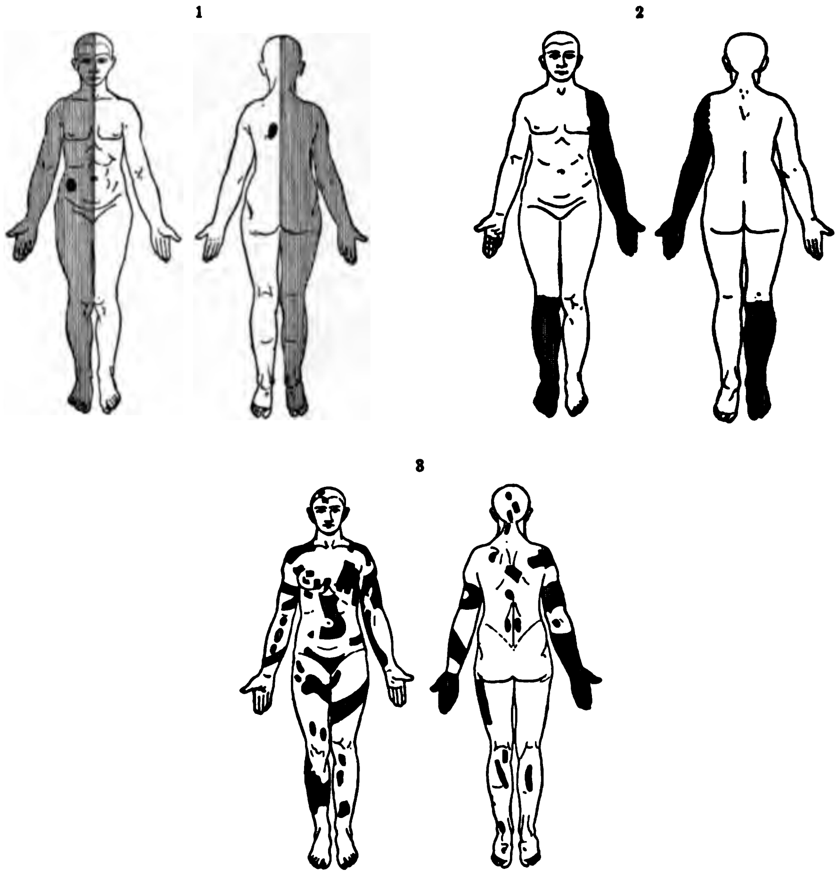

Fig. 249.—1, Most common disposition of hysterical anesthesia. Black spots represent hysterogenic zones (Dutil). 2, Hysterical anesthesia in geometrical segments (Tourette). 3, Hysterical anesthesia in disseminated islets (Pitres).

pharyngeal reflex is lost in ninety per cent. of hysterics. *Tickling* of the trunk or the *plantar* surface does not produce the usual responses. Buzzard lays especial emphasis on the loss of the plantar reflex, with retention of the knee-jerk, in hysteria.

In many instances the knee-jerk is relatively increased, and often a slight clonus may be obtained at the ankle. There is also a tendency for a tap on the knee to induce jerking of the trunk or both limbs, in addition to the true knee phenomenon.

3. The anesthetic zones are *movable.* Though they may persist for months, and even years, they are not absolutely fixed, and often are even capricious. A number of things may serve to promptly or gradually modify them. Hysterical attacks frequently change the distribution of the anesthetic zones, and sometimes are preceded by an increase in their size. During such attacks the anesthesia may completely disappear. Anesthetics or a dose of morphin or alcohol sometimes cause its temporary disappearance. Hypnotic somnambulism and suggestion may displace anesthetic areas, and they may disappear during sleep. If the patient's attention be strongly fixed on an anesthetic area, it may momentarily restore sensation. In the same way, as pointed out by Patrick, the boundaries of the anesthetic area may enlarge during the course of an examination, the attention in this case naturally being directed to the loss of sensation. The action of so-called *esthesiogenic agents* is supposed to be largely due to the attention and suggestion they invoke. By the application to the anesthetic areas of a great diversity of objects, such as metal plates, magnets, woods, metals, minerals, and gases, or by the use of electrical currents, especially faradism and static sparks, the anesthesia may be made to shift, sometimes to disappear, and often to *transfer* to the opposite side. Some patients are susceptible to one agent, some to another, apparently in proportion as their attention or fancy is captivated. When the anesthesia is transferred, it tends to return to the side whence it was displaced, but in doing so *oscillates* from side to side several times before finally locating in its old habitat. A *transformation* of tactile into visual sensation sometimes occurs. Binet first described this curious phenomenon. If letters, figures, or geometrical outlines be traced with the finger-nail or a pencil upon anesthetic skin areas, the patient can see them on a blank wall or screen. Fry [1] also found that if a colored screen were used, the patient described the tracing as surrounded by the color complementary to the one before her; for instance, with a blue screen the figures appeared to be in a red field.

4. It is a striking and paradoxical fact that hysterics are not disturbed by their sensory losses, and are usually entirely *ignorant* of them until deciphered by a medical examination. For instance, the small and delicate movements of the fingers ordinarily so dependent upon sensation may be perfectly performed by the anesthetic hand of the hysteric under the guidance of the eye. Apparently the motor image is called up and exteriorized through the motor paths.

5. It has been noticed that a hand completely anesthetic to all forms of sensation would yet grasp a pencil or other familiar object, and

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[1] "Jour. Ment. and Nervous Dis.," Aug., 1899.

promptly indicate its use, when the patient's eyes were bandaged and all sensation denied. In the same way an anesthetic leg and foot may cause no incoördination in walking. These paradoxical features have usually been attributed to deceit and simulation, but are presented by similar cases the world over without possibility of collusion.

6. The sensory disturbance is frequently located by topical or traumatic causes. The frequency of left-sided hemianesthesia is related apparently to the lesser strength of the left side. The superposition of sensory disturbance to inflamed joints or to injuries or to points of focused attention may often be noted.

Hysterical hyperesthesias are very common. Neuralgic pains and other disturbance of sensation may occur in hysterics as well as in others, without having any special significance, but often from such a source or from a blow or from the mere fixation of attention, perhaps through misdirected solicitude of the physician, there develops a *peculiar sensitiveness*. This is usually circumscribed. It may involve a joint (Brodie's joints) or an entire limb, but is practically never generalized or even of hemiplegic distribution. It is often confined to narrow, superficial zones or points, as at the vertex, giving rise to the classical *clavus*, about the breasts, along the spine, in the groins, and at the pit of the stomach. The glandular portions of the breasts, testicles, and ovaries may be similarly sensitive. Such sensitive spots are frequently found in the midst of anesthetic areas, and while the surface may be exquisitely sensitive to the touch of a finger, the pricking of a pin may cause no discomfort. It will frequently be found that if the patient's attention is strongly diverted, the area so intensely sensitive may be less sensitive than normal and entirely tolerant of firm and deep pressure. In the same way, by suggestion or the application of various inert substances, the sensitiveness may be, at least momentarily, suppressed. There is always found, upon close investigation of such sensitive areas, paradoxical peculiarities similar to those in the anesthesias, and which serve to proclaim their hysterical nature.

If such a hyperesthetic zone arises from or becomes associated with some mental storm, pressure upon it may serve to revive the memories in question and provoke a hysterical fit. It is then denominated a *spasmogenic* or *hysterogenic point* or *zone*. The mental character of hysterical hyperesthesias is evident in their genesis and associations. Of a similar nature are the disagreeable or painful sensations provoked in rare cases by certain substances or by heat or cold. Paresthetic tinglings, numbness, etc., are comparatively rare.

Motor Stigmata.—The motor stigmata of hysteria must be sharply distinguished from the motor accidents to be considered later. The stigmata are usually unknown to the patient, and must be sought by the physician. Practically, they differ from the accidents only in degree, and show the substratum of more marked errors of motor control, which are likely to develop as accidents at any moment. In many ways they are indicative of the automatism which is one of the most marked features of the psychoneurosis.

1. Movements in hysteria are *retarded*. The reaction time is in-

creased often for the simplest movement, and in proportion to the insensibility of the parts brought into play. The retardation, therefore, is closely allied to the anesthesias. It may be increased by diverting the patient's attention or diminished by concentrating his attention upon the given act. In automatic movements provoked subconsciously or under hypnotism the retardation disappears, showing its psychical nature.

2. Movements are maladroit and *incoördinate*, unless carefully supervised by the patient; and this, again, is proportionate to the anesthesia and the obliteration of the muscular sense. In automatic and subconscious movements the incoördination disappears. Partial or complete catalepsy and automatism in such limbs may be present if the patient can not see the limb or otherwise gain an idea of its position.

3. Hysterical patients are often incapable of performing several acts simultaneously, as they are unequal to the division of attention thereby necessitated.

4. Voluntary intentional movements are usually *weakened*. Patients who may in an automatic manner show great strength in the performance of customary and habitual tasks, when asked to make a test effort, as upon the dynamometer, register an insignificant amount. They appear incapable of willing an effort of which they do not possess the deeply graven motor image. The difficulty again is mental—the outcome of their diminished volition and attention. Amyasthenia is most marked in the insensitive parts, and foreshadows the paralytic accidents.

5. In many hysterics there is a tendency to rigidities or contractures. It may be demonstrated in weakened or anesthetic limbs, and is often indicated by exalted reflexes. It may be provoked by an elastic bandage, deep massage of the muscles, sharp flexion or extension of joints, faradization, frictions, percussion, and suggestion, varying in different patients. The contractures thus induced invade both flexors and extensors, and fix the joint in a characteristic attitude, the same as that in the accidental and spontaneous hysterical contractures. By similar means it can be caused to subside. Charcot denominated this state the *diathesis of contracture*.

Mental Stigmata.—The mental stigmata of hysteria consist in a belittlement of memory and of will-power. The amnesia is sometimes due to the lack of mental concentration,—the outcome of the volitional enfeeblement,—and the loss of will-power is frequently manifested in the impulsive acts and general want of self-control.

1. *Amnesia.*—The forgetfulness of hysterics accounts frequently for their uncertain and contradictory statements, and has often unjustly laid them open to charges of deceit. In some instances the memory loss is *systematized*,—that is, it embraces a certain group of related facts pertaining to some person or event, while other contemporaneous incidents are recalled. In the same way a group of motor images may disappear,—such, for instance, as those for walking or writing or for articulate speech,—and astasia, agraphia, and motor aphasia result. In this way the recollection of a certain person may completely drop out of their minds, or they may lose all the words of an acquired language. In other cases

the memory loss may be *localized;* that is to say, it embraces a given period of time. Frequently after a convulsive attack, sometimes in traumatic cases after the initial accident, there is a loss of memory for a variable period of time antecedent to the incident in question, or for a period both before and after the mental disturbance. Rare instances are recorded in which the amnesia has been *total* and complete for all acquisitions up to or during a certain period of life. Such patients begin again to speak and learn as children. In some cases acts and impressions are forgotten immediately. A book or a short story may be read repeatedly with full interest and appreciation, but is at once forgotten. In all marked cases of hysteria there is some blurring of recent memories.

The amnesias of hysteria are analogous to the anesthesias, and, like them, under the action of hypnotism or in subconscious states, may be demonstrated to be purely functional. The memory impressions do exist, and they can be revived. Recovered cases similarly regain the faculty temporarily lost. Out of localized amnesias a *double personality* may arise, as under similar circumstances the hysteric loses one group of memories and regains the other, alternating between the two.

Aboulia implies absence of will-power. It expresses the impaired volition of hysterics, which reduces their powers of mental concentration and attention, and renders them vacillating, impulsive, and lacking in determination. In some cases it attains such prominence that they can not bring themselves to undertake the simplest task, such as dressing or undressing, and hesitate at the slightest obstacle.

Impressionability or suggestibility is often extremely developed in hysterics, and practically constitutes a mental stigma. They are emotional and easily swayed, subject to the slightest influence and sensitive to insignificant impressions. ·The lack of will-power renders them of infirm and vacillating judgment, so that they often become dependent upon others to decide their slightest actions. The insistent, underlying, fixed idea thus controls them the more thoroughly.

Simulation.—From inadequate knowledge of the disease, hysterics have been supposed to simulate for the pleasure of it, and to deceive for the satisfaction derived from notoriety. To a very slight degree this may be true, but the simulation and the deceit have their origin in misconceptions, in misconstrued impression, or arise from the failure of impressions to reach the field of consciousness. Hysterical young women are too commonly supposed to be *erotic.* This is sure to be the case if in their attacks they show emotional attitudes or actions that may be so interpreted. As a matter of fact, they are usually frigid. Introspection and self-concentration are fatal to the grand passion. Local anesthesias or hyperesthesias may completely destroy the genesic sense.

ACCIDENTS OF HYSTERIA.

We come now to the more or less transitory features of hysteria, the accidents, chief among which are the hysterical attacks. These often take the form of severe, intense, and prolonged convulsions. While uncommon in this country, they are occasionally observed in the most typical form. Ordinarily, however, the attack is partial or larvated. Only by an understanding of the extreme variety will we be able to estimate the simpler forms, which usually consist of isolated elements of the typical *grande attaque* of Charcot.

Hysterical Convulsive Attacks.—The complete *grand attacks*, as studied and described by Charcot and figured by Richer, are infrequent, but in some *irregular* or fractional form they occur in the very large majority of all cases of hysteria. Patients possessing spasmogenic zones may usually be thrown into a convulsion by firm pressure on these points, and during the seizure similar pressure again commonly causes it to subside. Emotional disturbances may cause the attacks or they may apparently come on spontaneously.

The grand attack consists of a premonitory stage, followed by four periods: (1) The *prodromal stage* varies in different patients, but is uniform for the given case. Some patients are depressed, taciturn, and moody; others exhilarated, restless, quarrelsome, and talkative. Many have hallucinations of sight or of hearing, referred in the direction of the anesthetic side, and the insensitive areas may be increased. Usually, palpitations and vasomotor storms are observed. There may be nausea, hiccup, trembling, and the discharge of a large quantity of urine. The *aura* follows. This, ordinarily, consists of a painful feeling arising in the lower part of the abdomen, and develops into the sensation of a rounded body, which mounts upward, and, as it reaches the neck, gives rise to feelings of strangulation or suffocation—the *globus hystericus*. The face may flush, hissing is heard in the ears, throbbing is felt in the temples, objects turn dark before the eyes, vertigo occurs, and the patient sinks down, or even falls suddenly, unconscious, and the fit begins. In many instances the fit does not develop, and in a moment the patient is relieved, or the globus may last for hours without eventuating in a fit. Sometimes this train of symptoms is incited by emotional disturbance arising from insignificant irritation, or any strong mental impression may induce it.

2. *The epileptoid period* closely mimics the features of an epileptic attack. There may even be an initial harsh noise or slight cry. The patient is rigid; the face is pale, but promptly becomes red, and the neck is congested and swollen. Frequently the tongue is protruded or the teeth may be ground together. Biting the tongue and involuntary urination are uncommon, but do occur. Usually the convulsion is most marked on the anesthetic side to which the face is turned. The *tonic phase* lasts two minutes or less, and is often attended by slow, rigid movements of wide range, with notable extension of the feet and supination of the hands or movements of circumduction, unlike anything seen in epilepsy. The fingers are usually clenched over the thumb, which may protrude between them; the chest and abdomen are fixed and the body is rigid. This tonic phase is followed by a *clonic phase*, in which rapid, small oscillations begin in the rigid members and in the face. The suspended respiration reappears in broken, arrhythmical gasps and sobs, the chest and abdomen acting independently. Noisy movements of swallow-

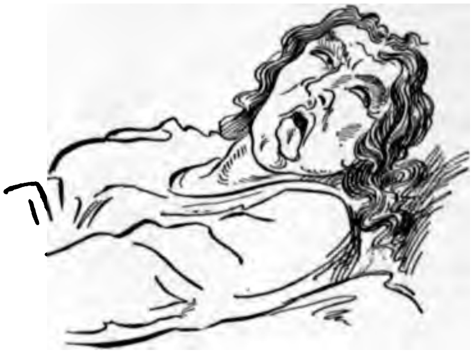

Fig. 250.—Tonic phase, the tongue rolling from one angle of the mouth to the other (Richer).

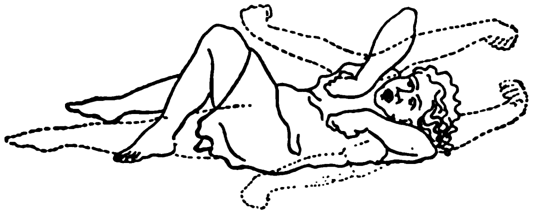

Fig. 251.—Schematic representation of the wide tonic movements (Richer).

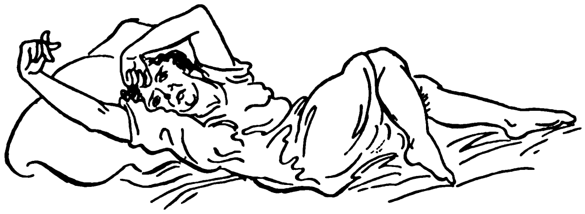

Fig. 252.—Tonic phase, circumduction movements of upper members (Richer).

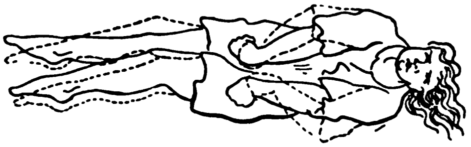

Fig. 253.—Clonic phase, schematic representation of clonic movements (Richer).

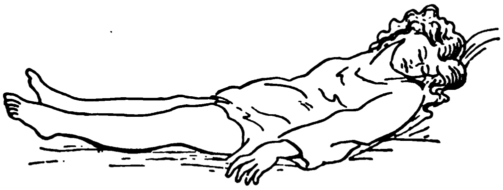

Fig. 254.—Phase of resolution (Richer).

ing and sonorous borborygmi are frequently produced. All the clonic movements are independent and illogical. This phase may last several minutes and gradually subside, the patient falling into a *phase of resolution*, in which some rigidity may, however, persist. Stertor and frothing saliva may appear, and sometimes general twitchings or sharp spasms

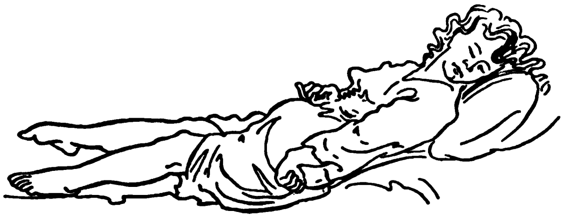

Fig. 255.—Phase of resolution, retaining partial contractures (Richer).

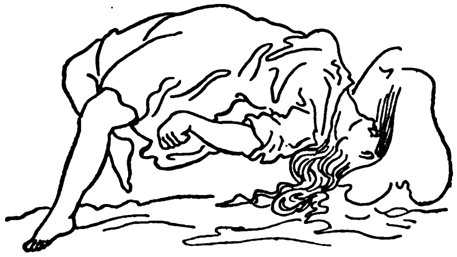

Fig. 256.—Posterior arc de cercle (Richer).

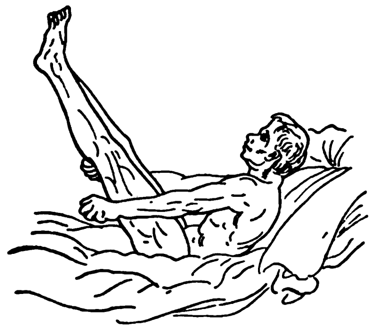

Fig. 257.—Anterior arc de cercle (Knobloch).

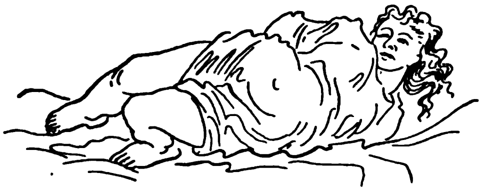

Fig. 258.—Lateral arc de cercle (Richer).

may still agitate the patient at intervals. The pupil may not respond to light.

3. *The Period of Clownism.*—After the phase of resolution of the epileptoid period has lasted a short time, the second period develops. It is made up of two phases,—(*a*) a phase of *contortions*, or illogical attitudes, and (*b*) a phase of wide-ranged, or *grand, movements*. The contortions of the first phase are usually steadily maintained for several minutes, thereby varying essentially from the grand movements of the second phase, which are repeated with more or less rapidity. One of the most common and characteristic contortions is an exaggerated opisthotonos, or *arc de cercle*. This may also be executed forward or laterally. To these contortions succeed alternate flexions and extensions of the trunk or of the limbs, or rotations of the head. This phase is attended usually by violent outcries, and, in evident fear or rage, the patients tear their garments, grimace in a horrible manner, and put forth an astounding amount of strength against those trying to control

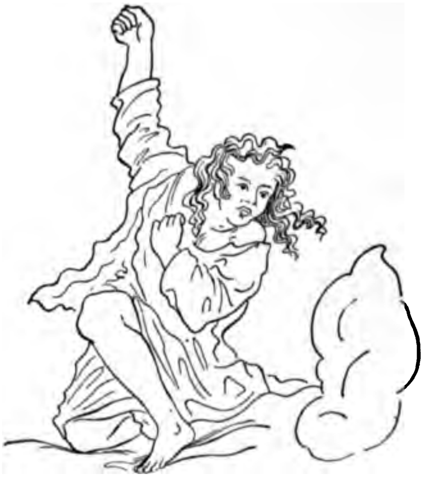

Fig. 259.—Passional attitude of struggling with an assailant (Richer).

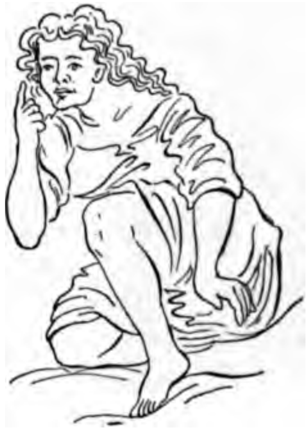

Fig. 260.—Passional attitude of solicitation (Richer).

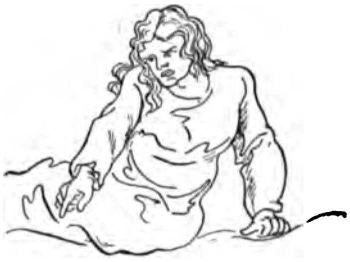

Fig. 261.—Zoöpsia (Richer).

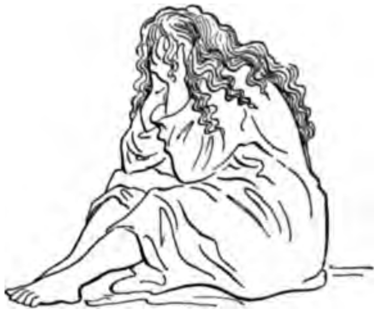

Fig. 262.—Delirium of the fourth period (Richer).

them. In this phase they often bite, scratch, and strike at their attendants, apparently under the domination of the hallucinations of a fixed dream or delusional storm.

4. *Period of Passional Attitudes.*—The third period is the gradual outgrowth and logical continuation of the second. The patient dramatizes in pantomime the acts of the dream that embraces circumstances of the past life, or perhaps the incidents connected with the origin of the hysterical condition. Terror, love, gaiety, rage, eroticism, singly or by turn, animate the features and compel the attending attitudes and gestures. The acts, emotions, and attitudes of one attack are usually repeated with fidelity at each succeeding crisis by a given case. If one knows the nature of the dream, it is easy to anticipate its manifestation.

5. *The period of delirium* is a prolongation of the dream state of the third period. It still pursues and dominates the patient, who now talks in the delirium and verbally expresses his hallucinations, which usually have to do with disagreeable sights, animals, and acts. The panorama, in which red often predominates, unrolls from the anesthetic side. The passional attitudes may be occasionally repeated. The delirium may be gay, furious, sad, or obscene, and the language and actions correspond. After a varying time the hallucinations fade, sad memories may recur, with sobs and tears, and suddenly, or after a few moments' silence, the patient arouses, a little fatigued, and at once fully recovers his former conscious state.

The *duration of a grand attack* is variable, but on an average requires from fifteen minutes to half an hour. The prodromal stage and the fourth period may be very long. The epileptoid period rarely exceeds three minutes; the second period ordinarily lasts five to ten minutes; the third is still longer. In some instances one attack follows another without appreciable interval, or some feature of the attack, as the stupor of the first period or the delirium of the fourth, may be prolonged for hours and days, constituting a *status hystericus.* The following table may serve to systematize the features of the grand attacks:

Premonitory stage.	Prodromes.	Mental disorders and hallucinations. Functional organic disorders. Motor and sensory disorders. Aura, globus.
Epileptoid period.	Onset.	
	Tonic phase.	Slow movements and tetanic rigidity.
	Clonic phase.	Small, quick movements.
	Phase of resolution.	Sopor, stertor.
Period of clownism.	Phase of contortions, arc de cercle.	
	Phase of large movements, salaams.	
Passional period.	Passional attitudes and actions.	
Period of delirium.	Delirium, hallucinations, zöopsia.	

Modified or Partial Attacks.—The typical grand attack may be infinitely modified. It may be intensified or reduced in severity, but, what is of more importance, it may be disaggregated, and almost any feature of it may occur alone as a partial attack. These partial attacks may also be indefinitely prolonged in a condition of status. Some of the most commonly encountered ones follow:

Vertiginous Attacks.—The premonitory vertigo, obscuration of vision, ringing in the ears, and sinking feeling may recur repeatedly, without other features of the attack.

Globus attacks are among the most common of the larvated seizures. The patient suddenly feels the discomfort at the throat, or it may slowly appear, and only disappear after a considerable period.

Epileptoid Attacks.—The attack may cease with the phase of resolution belonging to the first period, or, recurring at this point, strongly simulate the epileptic status. The differential diagnosis is often extremely difficult, the more so as epilepsy may affect a hysterical subject, and convulsive manifestations of both neuroses may alternate.

Tetanic Attacks.—The seizure may be limited to the first phase of the second period. The *arc de cercle* suddenly appears, and may persistently endure, or to it may be added the clownism, with wide movements, giving, for instance, salaam attacks. Rage, joy, and other emotions may then modify the movements and the attitudes, and demoniacal or maniacal attacks are presented, in which the patient may be extremely wild and unmanageable.

Attacks of ecstacy are manifestations of the passional attributes of the third period. With or without premonition or aura, the patient suddenly becomes motionless, and the face expresses astonishment, admiration, adoration, or stupor. Cataleptic features are often added.

Syncopal Attacks.—After an aura or some feeling of discomfort the patient relaxes and becomes inert and apparently unconscious. The hands may be clenched or a tremor of the eyelids may indicate the spasmodic state. Respiration slows, the face may be pale or slightly flushed, but the pulse is unaffected. This attack may be prolonged into the sleep attack.

Attacks of Sleep.—This lethargic or trance-like condition may follow several ordinary attacks, or some emotional storm may induce it. Its onset may be gradual, or sudden and apoplectic. The appearance is one of natural sleep, but careful search will often detect slight contractures, as in the eyelids, which do not easily yield to the finger, in the ocular squint, or the position of the hands and feet. Frequently there is a marked tendency to catalepsy, and the limbs retain any position passively given them. The pupils are sensitive to light, and, though general sensation seems abolished, it is only masked, as such patients frequently report all that has occurred during the sleep. The pulse-rate, ordinarily normal, may be diminished or increased. The respiration is usually shallow and reduced in frequency. The temperature may be normal, subnormal, or slightly elevated. Such attacks may last minutes, hours, or months, and terminate commonly by a convulsive attack, by laughter or tears, or a few delirious words.

Somnambulic Attacks.—Some patients, under a sudden impulsion, start on long flights or make considerable journeys, in which they may travel great distances and consume many days. Finally, they come to themselves in great surprise at being from home, and may have no recollection of the intervening events. In a succeeding attack they may recall everything that transpired in the first, and, by a repetition of such protracted attacks, build up a double existence or personality, or the morbid state may eventually completely displace the normal one. In lessened degree automatism and somnambulic attacks of short duration are not uncommon in hysteria.

Motor Accidents.—Paralyses and Contractures.—Both the paralyses and contractures of hysteria are foreshadowed by the stigmata of amyasthenia and the diathesis of contracture, of which they may be considered temporary exacerbations. They are frequently combined in a given patient, and even in a given limb. They may follow: (1) Convulsive attacks; (2) mental impressions or shocks; (3) traumatism; and (4) various morbid states. It happens that after a convulsive attack a hemiplegic, paraplegic, or monoplegic palsy supervenes and tends to persist for a considerable time. The other inciting causes mentioned act with less rapidity, and there is usually, even in traumatic cases, an intervening period of freedom from motor difficulties,—a period of meditation, as Charcot denominated it, or of autosuggestion,—lasting a few hours or several days. In addition, it is to be noted that there is no necessary relation between the severity of the physical exciting cause and the extent or completeness of the paralysis and contractures. They are relative, however, to the mental shock.

Paralyses of hysteria are commonly marked by a sudden emotional onset, or they may gradually develop after some such moral storm. Rarely do they abolish every movement in a limb, and close observation will usually detect slight evidences of voluntary or automatic motility. The antagonistic muscle-groups are equally affected and the limb is quite limp. There are vasomotor and trophic disorders in very exceptional instances only. The local temperature may be slightly reduced, and edema be encountered. Electrical and tendon responses are normal. There may be superficial or deep anesthesia or hyperesthesia. Variations of the extent and intensity of the paralysis follow various influences or occur spontaneously. Ordinarily, hysterical palsies terminate in complete recovery, but may last days or years. In some instances they are succeeded by contractures, or palsy and contractures alternate. In rare instances the paralysis is clearly ideational, in that it occurs only for certain groups of voluntary movements, such as those of writing or walking.

Hysterical contractures present loss of power, with persistent involuntary rigidity, without modification of the electrical or tendon responses. It is a rigid palsy. The affected limb is more or less rigid, the muscles are tense and firm, and the contractures persist during sleep, but yield completely during general anesthesia. Trophic disorders are rare, except in cases of several years' standing, when the muscles may be changed by actual fibrosis and permanent surgical deformities result. Ordinarily, there is superficial or deep anesthesia, or superficial sensitiveness. In slight cases the contractures may subside during sleep. Rare cases present exquisite sensitiveness and severe paroxysms of pain. Commonly the onset and termination are abrupt, but may be gradual.

Contractures, once developed, show a tendency to protracted duration. They impress characteristic attitudes upon the limbs. The arm is usually adducted; the elbow wrist, and fingers forcibly flexed. The hand, when alone affected, may be flexed into a fist, with thumb under or between the fingers, or the hand may be extended or held in the writing position. The lower extremity is commonly extended in all its joints. Rarely, the foot is dorsally flexed. The muscles of the trunk are not infrequently contractured, causing deviations of the spinal column mimicking scoliosis and anterior and posterior curvatures. The toes may be extended or flexed. Many of these attitudes are transiently represented in the hysterical attacks, as shown in the figures on pages 590 and 591, and in some sense hysterical contractures or palsies may be considered prolonged localized attacks, or as a hysterical status of fractional extent.

Some of the more common varieties of hysterical motor accidents follow.

Hysterical hemiplegia is most frequent on the left side and is usually incomplete, most affecting the distal portions of the limbs. Instead of walking with the rigidity of organic hemiplegia, the foot and leg are limply dragged along the ground, the advance being made by the sound side. Ordinarily, there is anesthesia of similar hemilateral extent or other sensory stigmata on the same or opposite side. Commonly the face escapes, and, when affected, the lower half is most implicated. Almost invariably limited contractures will at the same time be found, or they may predominate over the flaccidity or alternate with it.

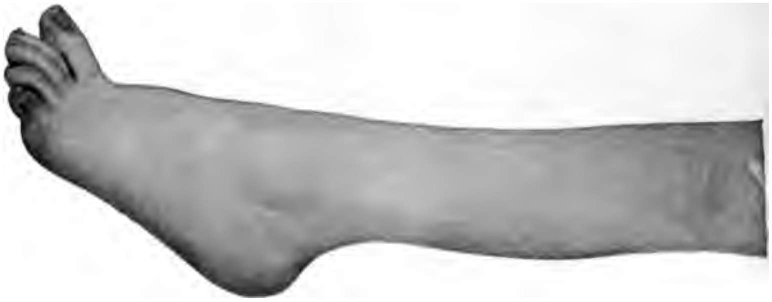

Fig. 263.—Hysterical contracture in foot and leg.

Hysterical monoplegias may be single or multiple. Their distribution may be unilateral or crossed. The face and limb on the same or opposite sides, or both lower limbs, or all four limbs may be affected. Usually the paralytic member is not affected in its totality, and presents an anesthesia of greater extent than the paralysis and of characteristic geometric outline. The paraplegic variety may disturb the urinary control, mainly through the anesthesia of the mucous surfaces.

The *face* may be affected either by paralysis or contracture. In the paralytic variety the upper portion of the face usually escapes, as in brain-lesions. In the spasmodic variety the lips and tongue are most affected, and an appearance of palsy on the opposite side is induced as the contracture drags the mouth to its own side. The spasm in the face and tongue is exaggerated by having the mouth opened and the tongue protruded.

Torticollis may be due to contraction or to paralysis, and the vicious position of the head corresponds. Contractures may be confined to the eyelids, giving a false appearance of unilateral or bilateral *ptosis*. The rigidity and resistance of the eyelids and the depressed eyebrows distinguish it from paralysis of the levator, in which the eyelids are relaxed and the eyebrow elevated.

Contractures and paralyses of the *ocular muscles* are seldom seen, but do occur. Convergent strabismus may appear. Conjoined movements may be impossible upon voluntary effort, but take place inadvertently. Divergent squint, palsy, or contracture of a single rectus or oblique are not seen in hysteria. Pupillary stasis is not an extreme rarity. Karplus [1] contends that in the majority of cases during hysterical attacks the pupil does not respond to light, and that this may also be the case in minor attacks.

Hysterical coxalgia may present all the attitudes of organic hip-disease, though abduction, outward rotation, and apparent lengthening are rare. The apparent deformity subsides under anesthesia, and the joint is found free and smooth. The joint also is not sensitive to percussion over the trochanter, or from the knee. Commonly there is an area of cutaneous sensitiveness, which is bounded by the iliac crest and Poupart's ligament above and a horizontal line below the trochanter. This may also be hysterogenic. Hysterical edema may mimic the local swelling and redness of actual disease, and, indeed, hysterical signs may be added to the true malady.

Astasia abasia is a hysterical condition characterized by abolition or disturbance of the coördinate movements for walking and standing In bed or on a chair the patient may show full coördinate control of the legs, but may be unable to stand unsupported, or, if able to stand, is unable to walk. Some such cases, while unable to walk, can run, leap, hop, or climb with ease. The difficulty is due, as already indicated, to a systematized amnesia.

Hysterical rhythmical spasms affect the limbs, face, or neck, causing movements, the same as those purposely executed in health, but steadily repeated with considerable force. They occur in attacks lasting from a few minutes or a few hours to several days. They cease during sleep, and apparently consist of a fractional part of a major attack. Among such rhythmical attacks may be named the *nodding spasm*, in which the head is nodded or shaken or rotated involuntarily, and so forcibly that it is impossible to check it by manual strength. The *choreic dance* or *saltatory chorea*, which played so important a part in medieval epidemics, is of this nature. Some patients move the arms as if using a hammer or other implement, and in general the rhythmical spasms show the dominance of a fixed idea. In a similar way the diaphragm may be affected, causing a peculiar, hoarse, *barking cough*.

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[1] "Jahrb. f. Psych.," 1898.

Sneezing or *grunting* may be repeated in more or less prolonged attacks. In very rare instances the movements may closely simulate the incoördinate, involuntary, arrhythmical action of Sydenham's chorea.

Hysterical tremors are of great interest, and often present very difficult diagnostic problems. In hysteria, and as an accident of hysteria, we may encounter all varieties of tremor, not excepting the intentional form that is so striking a feature of insular sclerosis. Hysterical tremors may be localized or generalized, fine or coarse, rapid or slow, intermittent or persistent, and may last for months and years. The tremor of Graves' disease, of paralysis agitans, of senility, and of metallic poisonings may be exactly counterfeited by hysteria. In some cases the tremor comes on in spells or attacks, in some instances it is limited to the anesthetic side or to an insensitive member, and in general its exact relation depends upon the escort of hysterical stigmata. The combination of hysteria with the organic diseases which are marked by tremor is very common. This is especially true of multiple sclerosis and the metallic poisonings. Charcot gave the following classification:

HYSTERICAL TREMORS.

A.	Tremors, not increased by voluntary movements.	1. Oscillating tremors, three to six per second.	Imitate paralysis agitans or senile trembling.
		2. Vibrating tremors, eight to nine or more per second.	Imitate Graves' disease.
B.	Tremors, occurring at rest or not, provoked or exaggerated by voluntary movements which do not accelerate their rapidity, but augment their amplitude.	3. Intention tremors, five to seven per second.	Imitate insular sclerosis or mercurial tremor.

Hysterical tics may be considered as fractional rhythmic spasms, occurring with more or less irregularity. Like tics in general, they have a purposive character, and are the expression of a fixed though usually subconscious idea. Winking, grimacing, shrugging, sniffing, coughing, movements of the hands, jerking of the legs, which may cause jumping, etc., are encountered.

Sensory Accidents.—Painful Accidents.—Owing to their more insistent nature the sensory accidents of hysteria are nearly all of a painful character. Sudden amblyopia and deafness have already been mentioned among the stigmata, but may appear transiently as accidents.

Hysterical cephalalgia may be deep-seated or superficial, in which case it is marked by a hyperesthetic zone. In rare instances it is most pronounced in the eyeball. It is likely to occur in periodic attacks, especially toward night, may be attended by vomiting and prostration, and is sometimes intense. It may be confounded with luetic headache, migraine, the pain of cerebral tumor, even with that of meningitis.

Hysterical pseudomeningitis is occasionally encountered and may deceive the elect. It may present malaise, loss of appetite, pains in the head, increasing to an insupportable intensity and leading to delirium. Vomiting, rigidity of the neck and extremities, fever, vasomotor streaks, and convergent squint may be added. Only a history of antecedent hysteria, an active pupil, a regular pulse, an absence of the dissociation between temperature, pulse, and respiration, and the presence of hysterical stigmata will enable a diagnosis if spinal puncture gives negative results.

Spinal irritability and tenderness are frequent, and sometimes constitute the dominant symptoms. The sensitiveness is exquisite, and may be localized over a few vertebræ, simulating Pott's disease, or extend the length of the spine. In many instances it is especially severe over the coccyx. The lightest touch, even the contact of the clothing, may be painful, and the patient can neither lean back in chairs nor lie on the back. Usually, if the patient's attention can be completely distracted, the sensitive zone is found tolerant even of vigorous, deep pressure. It is analogous to the hysterical hyperesthetic joints and localized hyperesthesias.

Visceral neuralgias are often marked by superimposed hyperesthetic stigmata, and may give rise to the suspicion of gastric or intestinal ulcer or malignant disease, or ape the crises of tabes.

Hysterical angina pectoris may exactly trace the features of stenocardiac attacks. In some cases it may attend an actual organic lesion, but usually there is no evidence of cardiac disease, and hysterical hyperesthesia in the precordial region is present. Such attacks are likely to occur in the night, and may have their starting-point in a dream. Often they are preceded or followed by emotional disturbance of a hysterical sort. Generally, the attacks are of short duration, but may persist for several days in a sort of status. The prognosis is not entirely favorable, in view of the fact that it is impossible to absolutely exclude an organic condition during life.

Visceral Accidents.—Respiratory Apparatus.—Associated with laryngeal paralysis and anesthesia, *aphonia* is encountered. It usually appears suddenly. The patient may whisper or execute low tones. Sonorous cough generally, and sometimes singing, may remain unimpaired, showing the ideational disturbance of vocalization and the systematized defect. Such patients may talk in their sleep with a voice of normal qualities. The laryngoscope may show the vocal bands unduly separated or approximated. *Mutism* is more clearly a cerebral defect, a systematized motor-speech amnesia. It may or may not be attended by aphonia. In rare instances *agraphia* has been present, but ordinarily the patient writes, gestures, and otherwise readily expresses ideas. The mouth parts usually are entirely mobile and free, but sometimes the tongue is contractured and can not be protruded. Hysterical stammering has also been recorded. Mirror speech is sometimes hysterical. The patient enunciates words backward. Repeated more or less rhythmic *cries*, *sobs*, *barks*, *hiccups*, *sneezing*, *laughter*, *grunts*, and various noises are met with, of which the type is the *hysterical cough.* They are often attended by sensations of a foreign body in the nasal chambers or pharynx, and appear usually in attacks occurring at various intervals, and presenting a variable duration. They cease during sleep. The cough is very frequent in hysterics of pubescent age, and may appear as the sole manifestation of the disease. A mental, moral, or physical shock may induce it, or abnormal conditions in the nasopharynx may be the starting-point. Acute inflammation or adenoids should be suspected.

Hysterical dyspnea may be (1) of sudden onset, due to laryngeal spasm; (2) it may result from contracture or paralysis of the diaphragm, and (3) it may consist of an extremely rapid breathing, reaching 60 or 100 respirations a minute. The laryngeal spasm is a rare accident, but is very alarming when it does occur, and has resulted fatally. Ordinarily, relaxation occurs when a certain degree of apnea has been reached. In the form marked by rapid respiration there may be no discomfort or effort, and the pulse is usually normal in rate and character.

Pulmonary congestion and hemoptysis are not very rare. Usually there is dullness at the apex, which may strongly suggest phthisis, but lacks the cachexia and temperature, and the sputum is free from bacilli.

Digestive Apparatus.—Hysterical *anorexia* sometimes reduces the patient to the lowest level of inanition. Patients reach such a degree of emaciation that they are practically living skeletons, and life is all but extinct. A fatal termination is quite possible. The origin of this state is in some fixed idea of suicide or of expiation. It may even arise from some trivial fear, as of growing too fat. In some instances an *esophageal spasm* causing *dysphagia* leads to the refusal of food, or it may follow vomiting. Hysterical *vomiting* occurs in attacks which may last days, weeks, and months. Food is simply regurgitated or violently expelled after a short stay in the stomach. Even fecal vomiting has been observed.[1] Sometimes there is *anuria*, and the gastric ejecta will be found to contain a corresponding amount of urea. The amount of vomiting and urinary excretion often alternate in close relation. In another form the esophageal spasm is temporary or more or less prolonged in the form of *globus*. *Tympanites* may appear in association with contracture of the abdominal muscles, and cause a *phantom tumor*, or, if attended by hyperesthesia, the appearances suggest *peritonitis*. Intestinal spasms are of frequent occurrence in hysteria, and may even produce prolonged obstipation by an *anorectal contracture*, which is often attended by severe pain and great sensitiveness. It may be mistaken for rectal stricture.

Urinary Apparatus.—In addition to the *anuria* and partial suppression of the urine attending or alternating with hysterical vomiting, and the discharge of large quantities of limpid urine of low specific gravity in the prodromal period of the attacks, or after any of the accidental crises of hysteria, *the amount* and *relative proportion* of some of the urinary constituents may be greatly modified. Tourette and Cathelineau found that the urine of hysterics in the interparoxistic periods of their disease was practically normal. Hysterical storms or temporary accidental crises, such as convulsive attacks and the various forms of hysterical status, like rhythmical spasms, delirium, or trance, presented a marked change in the urinary excretion. There is (1) a notable diminution in the fixed residue; (2) the urea is reduced about one-third; (3) the phosphates are decreased about one-half of the normal output for twenty-four hours. Upon examination they found (4) that the normal proportion of the alkaline to the earthy phosphates was altered. Ordinarily, it is in the ratio of alkaline 3 to earthy 1, but immediately after or during such attacks they become about equal. It is necessary to secure a twenty-four-hour collection on which to base such estimates, and it should embrace the period of the hysterical storm. Further, (5) in status lasting several days they observed that the diminution of urea lessened toward the end of the attack, and as it reached the normal amount the status yielded. Although any of the points may be presented in other than hysterical conditions and even in epilepsy, the combination of the first four may be taken as very characteristic. Many hysterics are troubled with *frequent urination*, which is especially aggravated if the mucous surfaces of the genitals or of the bladder are hyperesthetic. Mathieu [1] and Babinski assert that polyuria is generally, if not always, hysterical. In short, that diabetes insipidus is a hysterical manifestation.

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[1] Bregman, "Neurol. Centralbl.," Oct., 1901.

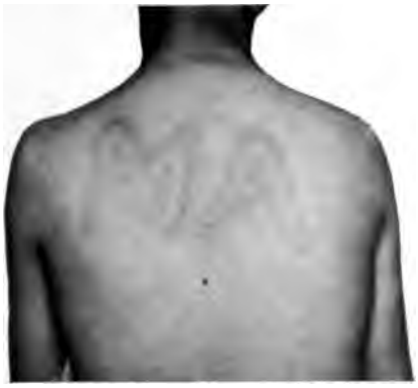

Fig. 264.—Dermographia in a male hysteric.

Hysterical fever may be continued, remittent, or intermittent, and usually, if not always, is found in female cases. After an interval of a few days, or perhaps only after many months, it may disappear suddenly. The temperature may attain a high range—105° or 106° are not infrequent, and 110° and higher have been recorded. Usually the physical state shows no corresponding febrile disturbance, but in some instances the tongue is heavily coated, there is headache, depression, sweats, and general phenomena of fever. Emaciation is very unusual. Commonly hysterical fever is unattended by other hysterical accidents, but it may be associated with pulmonary, meningeal, and peritoneal symptoms, and lead to mistakes. The intermittent form may suggest malaria. In the diagnosis of hysterical fever all sources of infection and hidden suppuration must be carefully investigated.

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[1] "Prog. méd.," Feb. 18, 1899.

Trophic and Vasomotor Accidents.—The trophic accidents of hysteria are of recent recognition. They are few in number, and are but rarely encountered in severe degree. On the skin *erythematous or vesicular eruptions* are the most common manifestation, and the vesicular form may go on to ulceration and produce persistent scars. Even *cutaneous gangrene* has been recorded. Cutaneous hemorrhages in the form of *bloody sweats* have followed severe emotional disturbances. In some historical cases they have given rise to the stigmata of the crucifixion or been attributed to the finger-prints of the devil. They are usually preceded by great pain in the parts. *Bloody tears* and bloody discharges from the mammæ or from the lungs are of a similar nature. Some hysterics show transitory periods of *dermographism*. *Neurotic edema*, which may be red, blue, or white, and *local asphyxias* similar in appearance to those of Raynaud's disease, may persist for many days, and usually appear in parts otherwise hysterically affected.

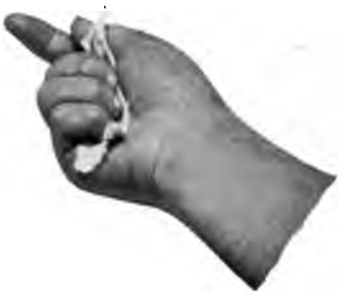

Fig. 265.—Hysterical contracture and edema of the hand, lasting several months. Index-finger not affected. Gauze used to prevent nails cutting into palm.

The *hysterical breast* is of rare occurrence, but furnishes a source of much apprehension and misapprehension. Suddenly, or within a few hours, one or both breasts enlarge, and are sensitive, painful, and hot. The skin may even be reddened. The nipple is turgid and sometimes erect. The glands are firm to the touch, but not edematous, and the hypersensitiveness is usually extreme. The enlargement may last several days or several months, and, if one-sided, may lead to suspicion of abscess or new growth. When bilateral, the condition has suggested pregnancy, especially as a milky fluid may ooze from the nipples.

Fibrotendinous contractures in muscles the seat of persistent hysterical contractures have already been noted. *Muscular atrophy* has been observed by a number of reliable observers. It has its seat in the muscles of a paralytic or contractured member, and rapidly develops. Within a few days the muscular masses may lose a third or a half of their volume. The condition then remains stationary for a long time, and finally recovers. There is a quantitative loss in electrical excitability proportionate to the muscular shrinkage, but the reaction of degeneration does not occur.

Course.—The course of hysteria is essentially chronic. Sensory stigmata have been known to persist for a lifetime, although accidents had long ceased to occur. It should be looked upon as a mental state which is likely to persist when once established, and as constituting a real disability, which may be partial or total. The various accidents may occur transiently, repeatedly, or persist for months and years, and must be individually considered in connection with each case. Under proper management hysteria is usually a manageable disease. Many cases get instant relief under certain mental and moral influences.

Prognosis in hysteria is clouded by the probability of recurrence under the action of inciting causes that otherwise might be trivial incidents. Many patients make substantial gain and consider themselves well, when an exhaustive examination discovers numerous persistent stigmata. These may be considered subjective recoveries. It is exceptional for well-developed cases of hysteria to regain absolute health. Children and youths make better recoveries than older subjects. Major hysteria after the age of forty presents very poor prospects of complete recession. Many of the accidents of hysteria and some of the stigmata are capable of instant disappearence, many of the disabling features are easily controlled and dissipated, but the fact remains that the curability of hysteria has been greatly overstated.

Diagnosis.—The diagnosis of hysteria loses many difficulties if it is clearly distinguished from neurasthenia, emotional disturbances, and wilful deceit with which it has been commonly confounded. They have nothing to do with hysteria properly considered, but may complicate it. While hysteria is polymorphous, and may mimic all other maladies, in that very fact lies its detection. There is in it always an excessive or paradoxical element. No disease, when well developed, is so distinctly marked and stigmatized. The most reliable and consistent features in hysteria are the mental characteristics and the psychic stigmata. Next in frequency and importance are the sensory stigmata, among which the disturbance of the color-fields by contraction and inversion is common and well-nigh pathognomonic. Anesthesias in islets or geometrical outlines are practically demonstrative of hysteria. The movability or motility of anesthetic areas under various influences is found in hysteria alone. Among the motor stigmata the contracture diathesis and the ideational loss of power for systematized movements, as shown in astasia or agraphia, while strength is normal in other respects, declare the hysterical state.

Some hysterical accidents are diagnostic. The typical hysterical seizure should be mistaken for nothing else. In the partial and much more commonly encountered seizures, attacks of ecstasy, of sleep, and of somnambulation are very distinctive. A careful examination of the palsies and contractures, taken with the ordinarily associated and usually superimposed sensory stigmata, should disclose their hysterical nature. The rhythmical spasms are the property of hysteria alone. The tremors must be deciphered from the context of hysterical manifestations and confirmed by the exclusion of organic processes. The same is true of the intestinal accidents. In the condition of the urine during and after paroxysmal manifestations we have a valuable index. Reduction in total solids, especially in urea and phosphates, with the inverted proportion of alkaline and earthy phosphates is, perhaps, only found in hysteria.

Once the suspicion of hysteria is entertained, a painstaking examination of the patient should confirm or banish it. The greatest danger arises from failing to appreciate the limitations of hysteria and allowing its presence to interrupt careful search for organic disease of which it may be a secondary expression. Too often, when hysteria is recognized, the physician is content to attribute every symptom and complaint to the psychoneurosis. Hysterics may have phthisis, Bright's disease, cerebral hemorrhage, typhoid fever, and hip-disease, as well as other persons. In every hysterical case the individual must not be forgotten or actual disease overlooked.

Treatment.—Recognizing in hysteria a mental disturbance principally, the treatment must be mainly psychic. This point of view may at once strengthen and weaken the physician. Unless he has a clear conception of the power of mental therapeutics, he is likely to look upon all measures as mere placebos and to lack faith in their value. Not confident himself, he fails to inspire confidence in the hysterical patient. The game is lost before it is begun. It is the self-confidence of the charlatan or the fanatical belief of the "Christian Scientist" that now sometimes succeeds when well-informed physicians fail. Expectant attention and hopeful anticipation have cured hysteria in all ages, and are potent measures to-day. Methods are usually successful in proportion as they are novel to the patient, strike the fancy, and stimulate the imagination. This serves in itself to distract the hysteric from the rut of his fixed ideas, and, if the assurance of help and cure is added and constantly repeated by suggestion, it tends to supplant the morbid condition. A great moral impression or a mental shock may terminate hysteria at once or may greatly aggravate it. Treatment may be considered (1) as *general*, applicable to all manifestations of the disease; and (2) *special*, regarding the manifestations of individual cases.

General Treatment.—The first consideration is, if possible, to *decipher the fixed idea* that dominates the patient. This is easily done when the hysterical syndrome originates in some serious mental storm or personal experience. In other cases it can be inferred from the hysterical manifestations, and occasionally it is constantly expressed in some worry or apprehension. In many cases, however, it is a subconscious idea. It may have arisen even in a dream, or it may be so intangible that it never is fully formulated in the patient's consciousness. Sometimes, from motives of shame, or modesty, or morbid conscientiousness, it is studiously concealed.

When once the end of the tangled skein is in the physician's hands, his task is to modify or *destroy the fixed idea*, and thus remove the source of morbid mentalization. Too often family and friends support the patient's morbid view and exaggerate the gloomy prospects, adding fuel to the flames by anxious solicitude and thinly veiled or openly expressed fears. In the highly suggestible condition of hysteria their constant presence and their consciously or unconsciously reiterated depressing suggestions counteract all possible good at the hands of the physician. The very locality in which the disease has developed constitutes a forcible reminder of its present and prospective woes. Unless the surroundings, companions, and visitors of a hysteric can be absolutely controlled, it is usually impossible to manage the patient. It is for this reason that *isolation* and separation from everything associated with the patient's morbid past is usually the first and most essential requirement of treatment. Under new circumstances the statements of the physician regarding the trivial nature of the dominant idea or his orders to dismiss it and his assurance of cure carry a weight and force that are not immediately destroyed by other more constant and less wholesome influences. The very fact of isolation is a profound influence that can readily be guided into a hopeful and helpful channel. A visit from an anxious mother or solicitous friend may, in a few minutes, destroy all advantage and recall the morbid past with added intensity. This plan of treatment can often be put in the form of the Weir Mitchell rest-cure, and requires the same conditions already indicated in the treatment of neurasthenia (p. 576). Mild cases, especially in the young, can sometimes be well managed by a long journey with a sensible and not too sympathetic companion, or by a protracted visit to friends or relatives properly informed of the attitude they should maintain toward the patient.

Hypnotism in its concentrated form is a dangerous measure and only of occasional service. In the hypnotic state the patient may readily disclose the hidden or subconscious idea, and it may at once be attacked and destroyed by countersuggestion. In the same way progressive improvement or immediate relief from the various conditions present in the patient may be suggested. Hypnotism may, however, precipitate a latent hysteria, and patients hypnotized for the removal of trivial hysterical symptoms have, in the hypnotic state or immediately after it, bloomed out in all the manifestations of major attacks or developed protracted paralyses and contractures. In other cases hysteria restrained by hypnotism has recurred with added force when the séances were discontinued, and Féré goes so far as to consider hypnotism but a transformation of hysteria. It should be held as a last resort.

It goes without saying that anemia and general states call for such remedies as are ordinarily beneficial, and local disease presents exactly the same indications as in non-hysterical patients.

Special Treatment.—The *convulsive attacks* can frequently be stopped by a dash of cold water, by a sharp command, by pressure on hysterogenic zones, and, if other means fail, by inhalations of ether. Their repetition depends upon conditions which must be met by the general measures previously indicated. *Paralyses and contractures* are among the most permanent accidents when once established, and become actual stigmata. If taken early, they can usually be managed. Massage, electricity, and repeated assurances of their early cure and of their insignificant importance is usually sufficient if the friends, in the way already described, do not defeat these measures. After a long duration they may require the full isolation treatment. Contractures of years' standing may be followed by fibrotendinous contractions only amenable to surgery. *Anesthesias and hyperesthesias* can be modified by a number of influences of the esthesiogenic sort. Faradic and Franklinic electricity often act very readily to reduce and completely dissipate the field of disturbed sensation. Various objects, metallic, wooden, etc., active or inert magnets, have the same influence. It is all a matter of concentrating the fixed and hopefully expectant attention upon the parts. In the same way these measures are useful in the palsies and contractures. *Spinal irritation*, so called, when once well marked, usually requires the isolation and rest treatment, but sometimes the actual cautery or flying blisters, or other heroic and hence impressive measures, succeed in removing the hypersensitiveness. *Aphonia and mutism* require treatment of a similar sort. A faradic electrode introduced into the larynx or pharyngeal cauterization has succeeded almost instantly in some cases, but lasting benefit usually follows persistent and repeated suggestion of steady improvement, coupled with various suggestive manipulations of the parts. *Laryngeal spasm* and hysterical *cough* or *sneezing*, or diaphragmatic spasms, generally can be interrupted by having the patient or a nurse forcibly pull on the protruded tongue. Laryngeal spasm in rare instances may require anesthesia or even tracheotomy, but, fortunately, apnea is commonly followed by relaxation of the spasm. *Dysphagia* from *esophageal spasms* is often relieved by passing the stomach-tube and demonstrating the permeability of the gullet. *Vomiting* is sometimes benefited by lavage of the stomach and the mechanical introduction of food, but assurance of improvement and suggestion must be added to all these measures.

Finally, it is the physician who is most sure of himself and of his diagnosis and has a distinct idea of the mental side of hysteria who best succeeds with general or special treatment.

CHAPTER VII.

EPILEPSY.

EPILEPSY has been the subject of medical description since the earliest times. It was known to the ancients as the sacred disease, *morbus sacer*. In colloquial English it is called the "falling sickness." Its most characteristic manifestation shows forth in the derivation of the name "epilepsy," which implies being seized upon. The seizing has been variously attributed to mythological deities, to the possession of devils, and to vapors and humors arising in the body. Focal epilepsy, or Jacksonian epilepsy, resulting from cerebral injury or disease, may be omitted from the present consideration. Epileptoid attacks arising from alcohol, lead, and uremia, and the eclampsia of parturients and children, have no necessary relation to the epilepsy now in question. Epilepsy can scarcely be considered a distinct disease. It is a syndrome of nervous and mental symptoms appearing under a variety of pathological states. In many instances it is associated with morphological cellular changes in the cortex. In as many or more cases the anatomical basis still escapes detection. So commonly is epilepsy presented by neuropathic and psychopathic strains, and in those physically or mentally defective, that in itself it may be considered a presumptive indication of degeneracy. It is statistically proven that there is more than one epileptic for every four hundred of the population in this country.

Etiology.—*Heredity* plays a very important part in the causation of epilepsy. Arthritis, syphilis, phthisis, inebriety, insanity, and neuroses are common in the antecedents. Epilepsy appears frequently in succeeding generations, and may descend directly from parents to children, but is more likely to be indirectly propagated by way of collateral branches. The heredity is most often by transformation from other neuropsychic disease. Thus, hysteria, epilepsy, and idiocy may follow in successive generations. Epilepsy among cousins is more frequent than among brothers and sisters, where, however, various neuropathic equivalents are frequently encountered. *Consanguinity* plays no part unless it brings together individuals of similar nervous or mental defect.

Epilepsy may appear at any *age*, but it is distinctly uncommon for it to commence after the age of thirty. Epileptoid attacks after that age should always awaken a suspicion of gross organic brain, heart, or kidney disease. Syphilis is the usual cause of such seizures from thirty to forty-five; after forty-five we encounter the degenerations of senility, vascular changes, and accidents. The very great majority of cases of epilepsy develop under twenty years of age, and the pubescent period, between twelve and seventeen, contains the greater proportion of them. Very frequently epilepsy begins in infancy. Convulsions during the first and second dentitions, incited by any febrile, septic, or toxic cause, may be followed by epileptic attacks at puberty. In some cases, beginning as eclamptic attacks in infancy or early childhood, epilepsy follows, with more or less periodical attacks from that time. Some families present numerous deaths from infantile convulsions, and epilepsy sometimes develops in those who escape. Nocturnal enuresis, pavor nocturnus, and epilepsy may appear in the same case, apparently replacing one another. It is necessary to carefully exclude from this consideration that large number of cases in which cerebral injury is present or brain defect arising from intra-uterine, birth, or postnatal causes. Such instances usually present evidence of cerebral lesions in some form of palsy, and are considered in part III. Males are somewhat more commonly affected than females, with a ratio of 20 to 16, according to Spratling.

Inciting Causes.—The alleged inciting causes of epilepsy, by their number and relative harmlessness in the majority of individuals, reinforce the presumption of a necessary predisposing defect, in the field of which they become active. The hereditary considerations, the usual evidence of degeneracy, and the common onset of the disease at developmental epochs all speak of a vicious organization unequal to the shocks of ordinary life and the demands of growth. In some cases, however, it is impossible to fix upon any predisposing state.

Toxic agents, such as alcohol, lead, mercury, tobacco, chloroform, ether, morphin, cocain, etc., have been accused of inciting epilepsy.

Alcohol is certainly competent to do so. The first fit may follow a drinking bout. Other *toxic conditions* arising from the intestinal tract or due to defective elimination through the kidneys may act as inciting causes. The *infectious diseases*, such as the exanthemata, may furnish the starting-point for epilepsy. *Typhoid fever* is causative in some instances. *Syphilis* may be causally related to epilepsy in various ways: (1) As a hereditary factor; (2) through the malnutrition of the secondary period; (3) by local encranial disease; and (4) Fournier thinks there is a peculiar syphilitic variety of epilepsy appearing late in the luetic history. *Head injuries* may produce focal epilepsy, but sometimes set up the common form of the disease. *Reflex irritations* due to nasal growths, intestinal worms, pelvic disease, genital abnormality or disease, masturbation, and eye-strain are occasionally sufficient to provoke epileptic attacks and to cause their repetition. Sensitive scars, decayed teeth, and ingrowing toe-nail have also been brought into this list. In fact, any local peripheral cause of constant nerve-fag may, in those of unstable organization, serve to upset the nervous apparatus. Physiological states, like menstruation and childbirth, may incite epileptic attacks in those predisposed. In female epileptics, the menstrual periods are very frequently the occasion of attacks. *Mental shocks*, particularly fright, may cause the first fit. Some epileptics have a fit upon the incidence of any emotional disturbance.

Pathology.—It would be impossible within reasonable limits to give the numerous theories that have been advanced regarding the nature of epilepsy, or to discuss at length the various locations assigned the morbid action resulting in the fits. Suffice it to say that the presence of mental symptoms, sometimes the limitation of the attack to unconsciousness alone, the experimental demonstration of cortical excitability, the usual organic basis of Jacksonian fits, the recent findings of changed cortical structures in some cases, and a recognition of the supremacy of the cell in the doctrine of the neuron theory, all combine to declare epilepsy a disease of the cerebral cortex.

It must be admitted that at present the most careful search fails in many cases to detect any histological changes known to be morbid. This is only another way of stating our ignorance of certain pathological processes and the limitations of present methods of research. When a patient dies as the result of repeated convulsions, venous and sinus engorgement, minute hemorrhages, and more or less extensive cerebral ecchymoses merely evidence the recent paroxysms. Lesions of longer standing have been frequently found, such as meningo-encephalitis, widely disseminated tuberous sclerosis in the cortex and the great ganglia, and diffuse sclerosis of the convolutions. Much importance was once attached to such hypertrophic sclerosis affecting the cornu ammonis or appearing in the bulb. Chaslin, Féré, and many others have repeatedly found neuroglial proliferation. Bloch and Marenesco have described vascular and perivascular changes in the cortex. Van Giesen has demonstrated changes in the large pyramidal cells of the cortex and in the neuroglia in two cases in which portions of the cortex were removed by operation. Bleuler,[1] in twenty-six epileptic brains, found a definite, wide-spread hypertrophy of the neuroglial bundles lying between the pia and outermost nerve-bundles. This change was not proportional to the severity or duration of the disease or relative to the mental degradation of the patients. In fifty-four non-epileptic brains similar changes were not found. Ohlmacher[1] draws prominent attention to persistent thymus and enlarged intestinal glands. Clark and Prout[2] describe wide-spread changes in the cortical cells, especially of the second layer, consisting of deformities, absence of chromatic substance, and swelling of the nucleoli with tendency to loss of the nucleolus under the section knife. Donath[3] shows that cholin is usually present in the blood, which, with other products of metabolism, such as ammonia and kreatinin, may act to bring about the attack.

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[1] "Münch. med. Wochens.," No. 33, 1895.

Symptoms.—The most striking symptoms of epilepsy are the "attacks" or fits and their modifications and equivalents. Of equal importance are the postepileptic states and the mental and physical conditions of epileptics.

Epileptic Attacks.—**Prodromes and Auræ.**—Many, but by no means all, epileptics have some distant or immediate premonition of the attack. Friends, nurses, and attendants upon epileptics often learn to anticipate an attack by temperamental or facial changes that are unnoticed by the patients themselves. These may be present several hours, or even several days, before the convulsive explosion. Forerunners of a motor sort are grinding of the teeth in sleep, twitching of the eyelids, face, or extremities, tremors, and restlessness. Some epileptics complain of tingling in the extremities or on the surface of the body; others have peculiar sensations on the tongue or in the nose, a tickling of the palate, or disturbances of smell or taste. Others have photophobia or brilliant muscæ. Ringing in the ears, genital excitement, respiratory oppression, vague discomfort, mental stimulation or depression, unusual irritability or gaiety, are some of the various premonitory conditions. Some patients look unusually pale; others present a suffused, puffy countenance; and temporary erythematous and urticarial eruptions have been seen. Much as these more or less prolonged premonitory conditions vary among themselves, they are comparatively uniform and constant for a given case.

The *aura* is the particular motor, sensory, or psychic feature that immediately announces the attack, and may be considered its initial symptom. In some instances it represents an abridged attack. It may recur for years unrecognized, until a fully developed fit indicates its true character. An aura of some sort occurs in fully one-half of all cases of epilepsy. *Motor auræ* may consist in a sudden limited muscular contraction or tremor, similar to the signal symptom of Jacksonian fits, but of only momentary duration. Automatic movement, and even a series of coördinate acts, may introduce the generalized attack. A patient scratches at the pit of the stomach, or winks his eyes, or moistens his lips with his tongue, or makes a few swallowing efforts, starts forward or backward, rotates upon his vertical axis, coughs spasmodically, springs up, or rolls his eyes to one side, following with his head and body. Unconsciousness at once follows, and the full fit occurs. Many epileptics have *sensory auræ.* A peculiar sensation is described, commencing in the feet or anywhere in the extremities, or in the abdomen, especially at the pit of the stomach. It mounts upward, and consciousness usually fails as it reaches the upper part of the chest or the neck and head. In some instances it strongly suggests the globus of hysteria. One patient describes it as a "nagging feeling," and it usually is of an indefinite and indescribable sort. Visual and auditory sensations are the most frequent of those that relate to the special senses. Those of taste and smell are very rare. Some epileptics hear certain sounds of a pleasant or startling sort; sometimes it is a distinct voice. Others describe everything as turning black or dark before their eyes; some have phosphenes or bright flashes; and in exceptional instances a definite picture is presented, such as that of persons or animals, which may rapidly approach or recede.

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[1] "Bull. Ohio Hosp. for Epileptics," 1898.

[2] "Bost. Med. and Surg. Jour.," April 23, 1903.

[3] "Med. News," June 28, 1905.

Jackson first called particular attention to the *psychic auræ,* which are as vague and varied as those of a motor and sensory character. A sudden recollection or the instantaneous review of a lifetime, a certain fixed idea or doubt, may announce the convulsion. Sometimes it is a rapidly growing mental depression, a fervor, a feeling of mental exaltation or weakness, irritable impulses, gaiety, rage, or quarrelsomeness.

Another group of auræ are called *visceral,* but are, for the most part, sensory or motor manifestations referred to the various organs. Precordial pain, violent cardiac palpitations, respiratory anguish, laryngeal spasm, weight at the pit of the stomach, vomiting, colics, and sudden imperative demands to evacuate the bladder or bowels are the principal ones. *Cerebral auræ,* such as sudden pain in the head, vertigo, stammering, and paraphasia, have been noted.

Lewis has noted an elevation of body temperature during the aura, and Voisin has observed increased temperature in the limbs in which the aura originated. Féré and Franck have noted an increase of arterial tension before the fit, and the writer has obtained a sphygmogram showing the same thing. All these considerations indicate that the aura is of cerebral origin, and serve to indicate the portion of the brain most at fault, just as does the original symptom in Jacksonian fits. They should assist in mapping out the functions of the so-called cortical areas of latent lesions.

Epileptic fits vary greatly in different cases, and usually a patient presenting severe convulsions also has slight or abortive attacks. We may consider (1) the complete attack, (2) the incomplete attacks, and (3) the equivalents of epileptic attacks.

The complete convulsion, preceded by an aura or not, comes on suddenly, consciousness and sensibility are instantly lost, and the patient falls. The fit presents three distinct periods: (1) one of tonic convulsion; (2) one of clonic convulsions; and (3) a period of stertor.

The *tonic stage* opens suddenly, with all the muscles of the body in tetanic rigidity. It results that the patient not only falls, but is frequently violently thrown down. At the same time, the sudden tetanic muscular grasp of the thoracic cage and the fixation of the laryngeal apparatus give rise to a cry that is practically never verbal. Sometimes it is a shriek; more often it is an expiratory guttural noise. The face is at first pale, but rapidly becomes congested and cyanotic, because respiration is inhibited. The eyes are fixed, often turned up or to one side or convergent, and the pupils are rigidly dilated. The dilatation is proportionate to the severity of the attack. Contracted pupils may sometimes be seen at the onset of the attack and again at the beginning of the stage of stertor. Minute vascular ruptures in the skin, mucous membrane, and conjunctiva often occur. It is probable that similar vascular accidents take place in the internal viscera and in the brain. If the hand be placed on the tonically contracted muscles, a thrill or vibratory impulse will be detected, such as is occasioned by excessive voluntary efforts. The tonic period is one of apnea, and only lasts one to two minutes at most. It may last only a few seconds. The tongue is frequently thrust between the teeth, and may be lacerated. The head may be slightly turned or retracted, the trunk is rigid and usually straight, the upper extremities, flexed at the elbows, are held close to the trunk, the fists are clenched, or the fingers may be made into a cone or spread by the interossei. The lower extremities are commonly rigidly extended. Complete symmetry of movements and position of the limbs is exceptional and quite frequently the lower limbs are partially flexed. It is at this stage usually that the spasm of the abdominal muscles expels the contents of the bladder, sometimes those of the rectum. The vascular strain is extreme.

The *clonic period* is in some sense a continuation of the tonic stage. The muscles relax, and again suddenly contract. The limbs, body, face, and all movable parts are agitated by the sudden sharp twitchings, which cause the face to grimace horribly, the jaws to grind together, often lacerating the tongue and cheeks, the eyes to jerk in their sockets, the air to be noisily forced in and out of the chest, churning the saliva in the mouth and throat and forcing it through the teeth and lips in a bloody foam. The arms, legs, and head beat against the ground. The clonic movements, at first rapidly repeated, gradually become less frequent, but maintain their force and vigor to the very last movement. The respiration, dominated by the clonic spasm, is inefficient; the asphyxia and cyanosis persist; the cardiac movements are precipitate; the arterial tension is intense. This stage lasts from one to five minutes, and terminates in full relaxation, in which the bladder and rectum, if not emptied in the tonic period, may allow their contents to escape. The body and face are covered with perspiration, the temperature mounts two or three degrees Fahrenheit. The clonic movements may be bilaterally synchronous, but have no suggestion of purpose, and are unmethodical and incoördinate.

The Period of Stertor.—The relaxation in which the clonic stage ends is usually marked by deep stertorous breathing and continued unconsciousness. The patient lies inert, just as the convulsion left him, profoundly comatose, and may perish at this time, smothered by the bedclothes or by similar mishap. After a few minutes, perhaps after a half hour or more, he opens his eyes and regains partial control of himself, without any knowledge of what has happened during the attack. If the attack take place during the night, the stertorous period may gradually merge into natural sleep, and the patient may be entirely unconscious of the occurrence of the fit. Usually the fit leaves the patient more or less confused, fatigued, sore, strained, and subdued for several hours or for a day or two.

While the typical fit presents all of these symptoms, it may be infinitely modified. The tonic period may be instantaneous only. The clonic movements may be confined to the face, upper extremities, or lower limbs. The cry, urination, stertor, or bitten tongue may any or all be lacking. The convulsion may be most marked on one side, and, indeed, exact symmetry is uncommon. The only constant feature is disturbed consciousness, but unconsciousness does not in itself constitute a fit, though it may practically constitute an equivalent.

Epileptic attacks often occur during the night, and in some instances only during sleep, giving rise to the clinical variety called *nocturnal epilepsy*, which may last for years without being suspected. Fits are likely to occur just as the patient is getting to sleep, or just as he is awakening in the morning. They may be induced by emotion, especially fright. Fatigue, coitus, menstruation, and child-birth may provoke them. In most cases, however, they take place without adequate traceable cause. Some patients have them very frequently, and with considerable regularity; others have them at irregular intervals; and others may have but two or three during a lifetime. In aggravated instances they occur in groups, several taking place within a day. Such groups frequently follow an interval more than usually prolonged, or succeed a period of bromid repression.

Occasionally, the patient, without recovering consciousness, has fit after fit, and in this way a series of hundreds may occur. This constitutes the *status epilepticus*, the gravest form of epileptic paroxysm. It consists of (1) a period of incessant convulsions, and (2) of a period of prostration and collapse; but sometimes death occurs in the convulsive stage. Usually, after the fits have continued for several hours, or even for several days, they become less severe, and finally cease, and collapse ensues. During the convulsions the respiration and pulse are accelerated and the temperature may reach 104° or 105°, and even rise higher, until death occurs. The individual fits of the status epilepticus are rarely of the severest variety. The stupor is punctuated more or less regularly by convulsive attacks of moderate force, and the status may even be made up of the limited attacks of the incomplete variety, such as vertigos, delirium, stupor, coma, cough, hiccup, and various psychic states. The collapse of epileptic status may be so profound as to reach a fatal ending.

Incomplete Attacks.—*Petit Mal.*—Fractional epileptic attacks present an endless variety, of which only a few of the common forms can be indicated. Almost any part of the full attack as previously described may alone constitute the limited seizure. Very frequently the disease presents an aura and the attack aborts. This may occur repeatedly, even for years, before the major seizures develop. Such warnings may occur in the intervals between the full attacks or after they cease, if the disease subside spontaneously or under treatment. A large number of cases present *vertiginous attacks*. The patient suddenly loses consciousness and falls, or nearly falls. There is usually slight twitching of the face, or perhaps only a little quivering about the eyes or mouth, or a deviation of the head and eyes, which may pass unperceived. Almost instantly the patient recovers himself, but may feel weak and faint. In other instances the patient falls heavily, but clonic movements and stertor do not develop. Again, the patient may fall, and become at once comatose and stertorous, as in apoplexy.

Of great interest are the attacks marked mainly by a *momentary loss or disturbance of consciousness*. The face may suddenly pale and the eyes look vacant. Whatever is being done is interrupted, and immediately resumed. The patient ordinarily has no knowledge of anything unusual having occurred. In talking he suddenly stops, looks absent-minded, and takes up the sentence where it was broken. A musician may pause one or two bars, and continue. At table the cup is poised half way to the mouth, etc. In other instances a little twitching of the face is noticed, or the patient drops or throws whatever he has in his hand, and usually there is momentary rigidity during the brief unconsciousness. In some instances a few words are muttered, or a little saliva dribbles from the mouth, and in such attacks the escape of urine is far from infrequent.

In rare instances the patient, under an irresistible impulse, rapidly walks or violently runs for a few moments in an automatic, unconscious way, and may fall finally in a fit, or may recover consciousness without an intervening convulsion; or after a fit a patient may suddenly run for some distance (*epilepsia procursiva*). Attacks of stertorous sleep have been recorded, into which the patient falls at intervals, and during which he can not be aroused. These may alternate with convulsive attacks or may be replaced by them. Attacks of generalized or localized trembling, local spasm, and salaam spasms may constitute the epileptic paroxysm. As before said, the only constant feature in epileptic attacks is a disturbance of consciousness during the period of the attack. Ceni [1] has observed sudden falling of body-temperature for about an hour, amounting to several degrees, sometimes recurring several times a day, and apparently constituting an epileptic equivalent.

Psychic Equivalents of the Epileptic Attack.—Instead of epileptic attacks of the ordinary kind, or in alternation with such, or as a repeated prelude to the major convulsions, or immediately following the fits, we may have a variety of acute mental disturbances. Epileptics may unconsciously, automatically, and with apparent purpose perform a number of coördinate acts. Homicidal, obscene, or pyromaniac acts may thus be done by epileptics, or intricate manœuvers, such as require the use of tools, may be accomplished. Subsequently, as a rule, they have no knowledge of such acts. The procursive fit may, in a sort of status, be prolonged so that the patient may make long flights, or, in a less violent way, make journeys of several hours' or several days' duration, during which the conduct is so natural as to attract no notice. Self-consciousness usually is rather abruptly restored, and they are astonished to find themselves at a distance from home, with an intervening blank period of time. Sudden wild, maniacal outbursts, in which the patient may be destructive and dangerous to others, are encountered, and these may terminate suddenly or be protracted for several days, attended by great excitement, a high pulse and temperature, and subsequent collapse. Sometimes such attacks are stopped by a fit, or they may succeed a severe convulsion, or they may take the place of the convulsion. Epileptic automatism may, in a static form, last days and weeks, during which the patient conforms naturally to his surroundings, but subsequently has little or no knowledge of the automatic period.

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[1] "Centralbl. f. Nervenh. u. Psychiat.," Oct., 1900.

Postparoxysmal Phenomena.—As physicians in practice rarely have the opportunity of seeing the patients in epileptic attacks, a knowledge of the postparoxysmal phenomena is important. As a result of the cortical exhaustion, a number of transitory symptoms follow the usual seizure. Tremor, paresis, disturbances of speech, of general sensibility, and of the special senses have sometimes been observed; the muscular strength is commonly reduced, the knee-jerks are diminished or abolished, and muscular tone is lessened. Onuf,[1] however, insists that during the convulsion and in the postepileptic period the reflexes are as commonly increased as diminished. Several attacks, repeated within a few hours, lead to an appreciable loss of body weight. The urine, even after a single attack, shows an increase of phosphates, particularly of the earthy phosphates, and of the nitrogenous constituents. The temperature is commonly subnormal. This may occur even in petit mal and has been noted in the brain itself by Mirto.[2] The toxicity and acidity of the urine are increased, and Agostini[3] finds the gastric juice has toxic properties. These properties are most marked just before and after a fit, and are proportionate to the intensity of the attack. Vomiting after an attack is not uncommon and almost invariably causes the fit to be attributed to errors of diet, which is sometimes the case. Cabitto[4] claims that the sweat of epileptics is hypertoxic previous to the fits, and urges the use of hot baths in the treatment of the disease, asserting good results from their employment. The special toxicity of the excretions in epilepsy is not proven. The arterial pressure is reduced, crises of polyuria, diarrhea, sweating, and salivation may be encountered. The presence of petechial ecchymoses, the bitten tongue, the voided urine, or fecal discharge is to be noted. Ordinarily, there is mental hebetude and sluggishness, a tendency to languor, and a desire to sleep. Headache is usually complained of, and often there is a feeling of extreme muscular fatigue and soreness. The psychic depression may reach a stuporous degree, and this may follow attacks that lack muscular violence. There is a decided tendency to mental deterioration, which, in the majority of cases, presenting frequently repeated major or minor attacks, markedly tends to dementia in the end.

Myoclonus Epilepsy.—In rare instances myoclonia and epilepsy occur in the same patient, constituting myoclonus epilepsy or the "*asso-*

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[1] "Am. Med.," Jan. 30, 1904.

[2] "Annali di Neurologia," 1899.

[3] "Riv. di Pat. Nerv. e Ment.," iii, 1896.

[4] "Riv. Sper. di Freniat.," 1897.

ciation disease." Such cases usually show marked degeneracy, and the disease begins early in life. Epilepsy usually antedates the myoclonic features which when once developed usually increase in severity and commonly eventuate in epileptic convulsions.

The myoclonus of the association disease is somewhat atypical compared with the essential myoclonias.[1] The contractions are usually lightning-like, but may have a fibrillary character, involving certain parts of muscles only,—a condition described as "live flesh,"—and such manifestations are likely to develop into typical myoclonic contractions, though they may remain fibrillary through the life of the patient. A single general tonic contraction may rarely constitute the entire clinical picture. Myoclonus contractions end imperceptibly in the tonic stage of the epileptic paroxysm. The contractions are often strong and affect large masses of muscles, rendering locomotion difficult. The trunk is frequently affected, causing the body to jerk backward and forward and laterally. The face and distal portion of the extremities are frequently involved. Generally the myoclonus is symmetrical, but both sides are not always synchronously involved. Commonly it begins in the upper extremities, and in a few days or weeks involves the lower extremities, the body, neck, and face in the order given, the muscles about the eyes and mouth being the last affected. The tongue and diaphragm frequently suffer, in severe cases producing grunts and barks, a sort of laryngeal tic. As the malady develops the myoclonus becomes more and more persistent during the waking state, having a tendency to develop the myoclonic status, which may terminate in death. As a general rule, superficial and deep reflexes are increased, and also, as a rule, there is lack of physical and mental development.

The prognosis as to recovery is extremely poor. Those who have reached advanced years present extreme senility and progressive dementia.

The diagnosis in a typical case is easy. There are family types and sporadic cases, the majority being of the latter variety. The condition may be acute and severe or mild and chronic.

The treatment can be only palliative, but, as a general rule, long remissions both in the myoclonus and the epileptic course may be obtained by the use of bromids.

The General State.—The bodily health in epileptics may leave little room for complaint. Very commonly they present gastric disturbances, a sluggish skin, and constipated bowels; but usually this is due to the bromids with which these patients are so commonly saturated. Many epileptics have gormandizing habits and insatiate appetites for food. This, with the inactivity often forced upon them by the disease and reinforced by the bromids, leads to flabby fatness. As a rule, they are indifferent and careless, and often the finer mental attributes and the keener sensibilities are dulled early in the disease.

Diagnosis.—The diagnosis of epilepsy is often very difficult, if the attacks are incomplete or not open to intelligent observation. The disease in the nocturnal form, or in the varieties of *petit mal* with slight attacks, may not attract attention for years. When the suspicion of epilepsy is aroused,—as, for instance, by bed-wetting, bloody stains upon the pillow, unexplained bruises, conjunctival ecchymoses, a dislocation or fracture occurring during sleep, cuts on the face or scalp, a bitten tongue, or some automatic or convulsive attack,—a careful investigation will rarely fail to disclose the nature of the disorder. In such cases a history of repeated momentary unconsciousness, or of some of the various sensory auræ, is significant. An account of nocturnal enuresis or nocturnal pavor in childhood is suggestive. Convulsions during teething, the marks of degeneracy, or the presence of a family neuropathic trace have some weight. If there have been repeated convulsions, and they conform to the epileptic type, presenting sudden onset, with or without warning, tonic, clonic, and stertorous stages, and complete recovery, there is little room for doubt.

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[1] Clark and Prout, "Am. Jour. of Insanity," Oct., 1902.

We have, however, to exclude the epileptoid fits symptomatic of various functional and toxic conditions. Intestinal, renal, metallic, drug, and alcoholic *poisonings* may cause convulsions that very closely, or exactly, copy the attacks of epilepsy, and they may induce epilepsy in those predisposed. *Vertigos* of gastric, aural, ocular, and neurasthenic origin may suggest *petit mal*. *Hysterical convulsions* are often mistaken for those of epilepsy. *Organic brain disease*, such as tumors, cerebral palsies in children, and brain injuries, may occasion epileptiform attacks. Finally, epileptic seizures may be of the apoplectic form and suggest *cerebral hemorrhage*, or the psychic equivalents may be confused with *transitory mania*. In every case it is obligatory to examine the patient rigorously from head to foot, both for the purposes of diagnosis and treatment. Of epilepsy, there is no one pathognomonic symptom. The following table shows some of the differential characters of epileptic and hysterical convulsions:

TABLE OF DIFFERENTIAL CHARACTERS OF EPILEPTIC AND HYSTERICAL ATTACKS.

THE FIT.	EPILEPSY.	HYSTERIA.
Prodromes.	Mental or physical premonitions.	Emotional disturbance.
Aura.	Common, but momentary.	Common and of considerable duration.
Onset.	Sudden, complete; cry, fall, rigidity.	Gradual.
Consciousness.	Instantly lost.	Partially lost or retained.
Course of convulsion.	Tonic, clonic, and stertorous stages.	Epileptoid and emotional phases.
Duration.	Two to five minutes.	A few minutes to several hours.
Positions.	Governed by flexors, mainly.	Tendency to extension, *arc de cercle*, opisthotonos, crucifixation attitude, etc.

Table of Differential Characters of Epileptic and Hysterical Attacks (*Continued*).

The Fit.	Epilepsy.	Hysteria.
Eyes.	Pupils dilated and rigid.	Pupils generally mobile and active.
Tongue.	Usually bitten.	Bitten very exceptionally.
Mouth.	Frothing common.	Frothing absent.
Sphincters.	Relaxed, usually.	Usually continent.
Pulse.	Accelerated greatly and tension increased.	Rate and tension not much changed.
Temperature.	Elevated 1° or 2° F.	Normal.
Termination.	In hebetude; gradual.	Rather prompt ending and little discomfort.
Urine.	Increased nitrogenous and phosphatic elements. Increased acidity and toxicity.	Urea reduced, phosphates decreased, and changed phosphatic formula; often large quantity, but of low specific gravity.
Postparoxysmal conditions.	Petechial ecchymoses, reduced muscular strength, diminished knee-jerks, mental obscuration; no memory of attack.	No motor or reflex changes; some recollection of phases of attack; usual mental condition at once regained; presence of various stigmata.

Prognosis.—As an exception, but proving the rule of gravity in the prognosis of epilepsy, rare cases recover spontaneously. In a general way there is little hope for cure, if the disease, with repeated attacks, has lasted over two years, or been intense in its manifestations for even a shorter period. There is some tendency for it to appear, subside, reappear, or increase at the developmental epochs of dentition, puberty, adolescence, and the climacteric. Any inciting cause that is controllable is a favorable consideration. Epilepsy as a manifestation of degeneracy is of bad import. If there is any reason to suppose that organic cortical changes have occurred, or if dementia has appeared, the outlook is unfavorable. Grand mal cases offer a slightly better prognosis than petit mal or psychic cases.

A single attack is almost never fatal of itself, but a condition of status is very likely to terminate fatally, and every attack undoubtedly leaves some harmful trace. The natural termination of epilepsy is in dementia. The oftener the attacks occur, the worse the mental prospects. Death by status, or through vascular accident, or by suffocation, or other physical mishap attending a fit, is not so very rare. Tuberculosis is a frequent cause of death. Epilepsy, with fits at long intervals, particularly if first appearing after twenty years of age, is not inconsistent with mental qualities of a high order, and may not shorten life or abridge usefulness. The majority of epileptics, under proper conditions, may be advantageously and profitably employed in colonies provided for the purpose.

Treatment.—A systematic study of the patient is the first step toward treatment. Every possible source of *local irritation* must be investigated. Occasionally, the aura may direct attention to some body-disturbance that acts as an inciting cause of the fits. Asthenopia due to errors of refraction, or faulty eye muscles, nasal disease, pharyngeal tumors, maxillary or dental faults, gastric inadequacy, intestinal parasites, rectal ulcers, hemorrhoids and fissures, constipation, diarrhea, vesical and genital troubles, must all be sought and corrected, if found. *Toxic conditions* are equally important. Auto-intoxication from the stomach, intestines, or kidneys must be stopped. Alcohol, lead, tobacco, coffee, tea, and dietetic errors may incite the fits in those predisposed. *Hygienic regulations* are of the greatest importance. These pertain (1) to the diet, which should be nourishing, easily digested, and of reasonable amount. Milk, fish, vegetables, and suitable fruit, with a small amount of starchy articles and a very little meat, is generally best for epileptics. In some instances an absolute milk diet is decidedly helpful, and may be continued for many months with advantage. (2) Baths and exercise to keep the skin, muscles, and circulation active, and out-door life to further the same end, are valuable. Often vigorous manual labor is advisable. (3) The rooms, ventilation, clothing, occupation, amusements, and habits of the patient are worthy of full consideration. Do not overlook the tendency to constipation.

Improvement under such measures is the rule, and in rare instances it is one's good fortune to see epilepsy subside after the removal of some irritation or the correction of some toxic state.

Turning to drugs, the most importance attaches to the *bromids*, but they should be reserved as a last resort or as an adjuvant. Cures by the bromid treatment are not to be expected. It is at best a palliative treatment and one often fraught with great disadvantage. Usually, in order to completely suppress the attacks an amount of bromid is required that maintains constant hebetude. As a practical fact, if the fits are not controlled by sixty grains of bromid a day, the question is quite sure to arise whether the epilepsy or the bromid is the greater evil. It is a frequent experience to see patients, brutalized by bromid, go months without fits, but with a loss of mental and physical activity. Sometimes the attacks then recur with seemingly additional violence, or status may develop. Among the bromids there is little choice, but the sodium salt is least likely to disturb the stomach. During the use of the bromids the intestinal tract must be kept aseptic by the use of large quantities of drinking water or some of the laxative waters, and the administration of some antifermentative, like salol or betanaphtol. If the fits are nocturnal, the medicine should be given after the evening meal; if they occur on rising, the largest dose should be administered at bedtime. When menstruation aggravates the epilepsy, the doses of bromid may be doubled for a few days before, during, and after the period.

Sometimes the use of antipyrin, phenacetin, trional, or other of the coal-tar derivatives, with bromids, gives better results than the bromids alone. Digitalis or belladonna in similar association will be found valuable in those cases where there is weak circulation. Sumbul and solanum have their advocates, but so has had every other remedy ever known to man. Flechsig has proposed the use of opium, to be followed by bromids, in cases of long standing. He gives opium in doses gradually increasing from one to fifteen grains or more a day, if well borne, and after a few weeks abruptly stops the opium, substituting bromid, twenty to forty grains, three times daily. Bechterew combines bromid, adonis vernalis, and codein with favorable results. Any one of these plans may be tried when bromid alone fails or loses its force. Richet and Toulouse[1] called prominent attention to the asserted value of withdrawing common salt from the food of epileptics, thereby securing, it was thought, a better absorption of the bromids. A very general trial of this measure has led to the belief that it is one of decided value, and as a rule smaller doses of bromids are more efficacious under this régime than larger ones without it. Even alone, without the use of bromid, the withdrawal of salt secured a reduction of the number of fits in fourteen cases reported by Garbini, and the attacks were less severe. The plan is entitled to a trial in all cases, but is often very difficult to carry out in the usual run of private practice.

CHAPTER VIII.

MIGRAINE.

MIGRAINE is an explosive, paroxysmal psychoneurosis. The attack, usually commencing with sensory and mental symptoms, is almost always attended by headache, which is frequently one-sided, and there is generally nausea and vomiting. It is sometimes called *hemicrania, sick headache*, or *megrim*. Owing to the vomiting, it is often erroneously attributed to biliousness.

Etiology.—*Heredity* is often strongly marked. It is more commonly direct than in almost any other neurosis. Migraine may sometimes be traced through several generations, numbering dozens of cases in a single family tree. Any neuropathic family is almost sure to present cases of migraine. It seems capable of transmission by transformation, alternating with hysteria, epilepsy, and insanity. It may be associated with the graver neuroses, or with psychoses, in a given patient. Gout and *arthritism* have similar close relations with it. Thirty per cent. of cases begin between five and ten years of *age*, and the balance appear mainly at puberty, adolescence, and during early adult years. In rare instances it may begin after thirty. The female *sex* is somewhat more commonly affected than the male.

The *inciting cause* is often obscure. Some cases date from periods of *lowered physical health* arising from any cause. The cases beginning in early childhood very frequently follow the first systematic use of the eyes for near vision, as in school-work. *Eye-strain*, arising from accommodative or muscular asthenopia, is certainly competent to incite migrainous attacks in those predisposed. Gouty or *lithemic* conditions,

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[1] Richet and Toulouse, Paris Academy of Sciences, Nov., 1899.

constipation, indigestion, fatigue, lactation, emotional disturbance, or any *febrile movement* may set up the attack.

Symptoms.—The symptoms of migraine are those of the attacks. These vary considerably in different patients, but are tolerably uniform for the given case. There are usually: (1) Premonitory symptoms; (2) sensory disturbances; (3) headache; (4) nausea; (5) vomiting; (6) sleep; and (7) complete recovery, occurring generally in the order given. In addition, there are usually vasomotor symptoms and occasionally mental and motor phenomena.

The *premonitory symptoms* are most common in the cases in which the early sensory symptoms are least marked. For a few hours or a day the patient feels heavy, dull, apathetic, and is usually indifferent and irritable. There may be slight headache or somnolence. After a nap he may wake up with a fully developed one-sided headache, or this may be present on awakening in the morning. The *sensory symptoms* occur in over half of the cases. They usually begin quite suddenly. Bright spots before the eyes, colored rings, luminous zigzags, hemianopsia, dimness of vision, clouds, etc., are some of the subjective visual disturbances. They affect both eyes, and are sometimes lessened if the eyes are closed. They are usually most pronounced or entirely confined to the homologous half fields. Some patients only feel a vague ocular discomfort, or decided photophobia may be present. Taste and hearing are exceptionally affected in a similar manner. In some cases there are sensory symptoms in the limbs, face, throat, or tongue, but especially in the hand or foot. A tingling or numbness invades the parts and gradually advances toward the center. These sensory disturbances usually last ten to twenty minutes and then subside, the headache immediately displacing them.

Motor symptoms, though exceptional, are very valuable indications of the cerebral nature of this neurosis. The extremity which presents tingling may show paresis, and the following headache is usually on the opposite side. Motor aphasia may be added to right-arm tingling and left hemicrania, and the left halves of the retina may be disturbed by visual sensations, which are referred outwardly to the right fields. Temporary word-deafness has also been recorded. In another group of rare cases there is transient unilateral paresis of the oculomotor, marked by ptosis, outward squint, double vision, pupillary dilatation, and loss of accommodation, constituting the so-called *ophthalmoplegic migraine.*

Slight *mental changes* occur in some patients, such as depression, mental confusion, restlessness, loss of memory, stupor, double consciousness, or a recurrence of some vivid memory.

The *headache* is the most uniform, dominant, and distressing symptom. It varies in different cases in degree, duration, and location, but is commonly intense and ordinarily circumscribed, at least at first. Often commencing as a localized, intense pain in a small spot in the temporal, frontal, ocular, or occipital region, it gradually spreads to the rest of the same side of the head, or may become diffused all over the head. Less commonly it commences on both sides as a frontal or occipital pain. Rarely, it passes down the back of the neck and into the arm. The character of the headache is tolerably uniform in the same case, but some patients have several varieties, which reappear from time to time, and are recognized as old acquaintances. The headache lasts from one or two hours to ten, twenty, or forty, and may subside abruptly after nausea or nausea and vomiting, or gradually grow less and disappear. During the height of the headache the patients usually shun light and noise, and remain as quietly recumbent as possible. Movement, such as rising or stooping, intensifies the pain. Tenderness of the scalp or nerve-trunks is unusual.

In most cases *nausea* appears after the headache develops or has reached its height, and there is complete anorexia. Digestion appears to be stopped, as unchanged food is sometimes vomited many hours after its ingestion.

The nausea leads to *vomiting* in a fair proportion of the cases, and emesis is attended by much retching and difficulty. It is often repeated and protracted, so that biliary matter may finally appear in the ejecta. Usually, once commenced, it is provoked by swallowing any fluid, or even by the saliva, which is commonly apparently increased in amount. Often the patient is cold, pinched, clammy, and suggests the collapse of seasickness or choleraic disturbance. Frequently, as the vomiting subsides, a feeling of great relief is experienced, the headache ceases, and the patient falls into a quiet sleep of a few minutes or several hours, from which he arises and asks for, or tolerates, a little food.

The *vasomotor symptoms* are interesting, and have attracted great attention. Early in the attack, before the headache has appeared, there is frequently pallor or mottling of the face. In some instances a vivid red streak in the middle of the brow or a one-sided flush invariably appears. The pallor is succeeded by flushing, in some, and there may be general profuse perspiration. Commonly, the extremities are cold during the severe pain, the pulse sharp and retarded. Usually the contracted pupils show the participation of the cervical sympathetic. This rarely is unilateral, and may produce retraction of the eyeball. The inhibited digestion may be due to a similar angiospastic condition of the gastric arterioles. As the attack declines, the surface reddens, the pulse resumes its proper rate, the pupils relax, the pallor disappears, and in rare cases some puffiness in the scalp has been noted. Increased diuresis may follow. In the intervals the patient may feel perfectly well.

Course.—Migraine has a tendency to persist for many years, when once established. Commonly, in women, after the menopause, it subsides, and it disappears in men after fifty-five or sixty. The attacks occur with more or less regularity, and sometimes with remarkable periodicity. Menstruation may provoke it monthly in women. It sometimes occurs every Sunday, especially in men who change their daily routine at that time. Irregular intervals of weeks or months may intervene, when any of the inciting causes may precipitate it; but it is usually noticeable that an inciting cause or condition, acting soon after an attack, fails to induce an immediate recurrence. It is evident that the attack has cleared the atmosphere and exhausted the susceptibility. In certain rare instances the migrainous attacks have been replaced by epileptic seizures presenting the same premonitory features. A case has been seen presenting migraine, epilepsy, and transitory mania, apparently as alternating equivalents. Krafft-Ebing[1] reports a number of cases in which transitory mental disorder occurred as part of, or in alternation with, migraine. The premonitory hemiopia in a case reported by Noyes became permanent. In advanced years the migraine may apparently be replaced by labyrinthine vertigo. After many attacks, some intellectual impairment has been noted. Granting the neuropathic substratum in migraine, the association or succession of other neuroses and psychoses is surprising only by its rarity.

Pathology.—In the absence of knowledge regarding the morbid anatomy of migraine, we are thrown back upon theories and analogies. Attracted by the vasomotor symptoms, many attributed the migrainous attacks to disturbance of the sympathetic. This is a clear confusion of effect and cause, of symptom and disease. Taking into consideration the cortical features manifest in sensory disturbance, hemiopia, tingling, aphasia, motor loss, crossed hemicrania, mental features, cardiac and digestive inhibition, and the vasomotor disturbance itself, there can be little doubt that migraine is a cerebral disorder. Its resemblance to epilepsy, if not its actual relationship,[2] points to the same conclusion. The exact nature of the cortical instability is for the future to reveal.

Diagnosis.—The diagnosis of migraine depends mainly upon its paroxysmal and recurrent character and its definite clinical features. The sensory premonitions and vasomotor phenomena are very significant when present. In cases presenting migrainous attacks of the milder sorts, it is difficult to be sure that the headache is not due to some ordinary cause, until its repeated recurrence under somewhat similar circumstances declares its nature. "Sick headache" is almost invariably migraine. The vesperal headaches of *syphilis*, and the quotidian or tertian headaches of *malaria*, occur with greater periodical regularity and with much shorter intervals than the attacks of migraine. Both lack the nausea, vasomotor symptoms, and complete recovery. From *petit mal* the diagnosis may often offer considerable difficulty. The premonitory sensations may be taken for an aura, but their prolonged duration is unlike the momentary warning of epilepsy. Unconsciousness does not occur in migraine; it is the most constant feature of epilepsy. Headache, as a symptom of other disease developing in a migrainous patient, may be overlooked and the concurrent malady neglected. The headaches of Bright's disease, of cerebral tumor, of syphilis, or malaria may be wrongly attributed to the neurosis.

Prognosis.—Migraine is usually a stubborn and persistent malady. It has a tendency to last until involutional changes in the organism commence, when it frequently spontaneously subsides by a gradual increase of the intervals between the attacks, rather than by a lessening of their severity. If the disease is of short duration, and some remov-

[1] "Alienist and Neurologist," Jan., 1900.

[2] Wilfred Harris, "Transient Hemiopias," "Brain," 1897.

able cause can be discovered, the prognosis is fairly good. In older cases the attacks can usually be rendered less frequent and often aborted if the patient is watchful and persists in treatment.

Treatment.—Unless the condition or agent provocative of the attacks can be discovered and removed, there is little likelihood of fully successful management. To this end all sources of peripheral irritation and auto-intoxication must be carefully investigated. Eye-strain, improper diet, excesses, or bad habits of any sort must be corrected. In some migrainous patients any relative excess of nitrogenous food is sure to induce an attack. As a rule, for these patients an abundance of outdoor air, free cutaneous and intestinal excretion, and an unstimulating diet are indicated. A tablet of $\frac{1}{100}$ of a grain of nitroglycerin, allowed to dissolve in the mouth, and taken at the earliest premonition, will sometimes abort an attack. Caffein has a similar effect with some patients. Others, by taking a large dose of bromid and lying down, occasionally escape. Others, again, by inducing emesis, or by washing out the stomach, interrupt the paroxysm. When the attack is once on, heat or cold to the head, warmth to the extremities, and a mild sinapism over the stamach are helpful. A dark, quiet room is usually demanded. Morphin will control the pain, but should, if possible, be avoided, to prevent setting up the opium-habit.

If the attacks are of considerable frequency,—say one or two a week,—a continuous course of bromid, as in epilepsy, may give good results. In the forms marked by paretic symptoms of onset—by ophthalmoplegia, for instance—the bromid treatment is of the greatest value, and such cases should be managed much as cases of epilepsy with incomplete attacks.

CHAPTER IX.

NEUROSES FOLLOWING TRAUMATISM.

THE subject of neuroses following injuries is one of vast importance to the general practitioner, and has a medicolegal side of much interest. The question has been greatly befogged for many reasons. The first important work directing attention to this class of cases was the publication of Erichsen in 1871 on "Spinal Concussion," reprinted in 1875. In it he reports fifty-three cases resulting from injuries, received mainly upon railways. These cases present all manner of lesions, alone having in common the negative feature—absence of external evidence of injury. In this list are embraced cases of fracture of the dorsal vertebræ, hemorrhage into the cord, division of the cord, meningitis, simple nervousness, hysteria, neurasthenia, and pretty much everything else. Unfortunately, "concussion of the spine," as set forth by Erichsen, was seized upon by lawyers as the basis for prosecuting suits for damage against corporations, and it became fixed as a disease-entity in the legal and medical mind. In 1883 Page, a railway surgeon, brought out a book written from an *ex parte* standpoint to counteract the contentions of Erichsen, and the battle raged fiercely from both sides for a number of years. Subsequently, in Germany, Oppenheim, in 1889, made a closer classification between organic injuries and those not marked by gross histological changes, and proposed the term "traumatic neuroses" for the latter. About the same time, and subsequently, Charcot taught and demonstrated that the nervous symptoms in these cases, apart from those attributable to organic lesions, were precisely the same as are presented in neurasthenia and hysteria.

The early errors of Page and Erichsen are easily understood if it is recollected that the finer anatomy of the nervous system, as well as the physiology of the cord, was but little known in their time; that the electrical reactions of nerves and muscles were not clearly understood; that the stigmata of hysteria and the symptom-group of neurasthenia were still undeciphered. Unfortunately, there was a tendency to dignify all the morbid conditions following serious injury by such terms as "spinal concussion," "railway spine," "traumatic neurosis," and "Erichsen's disease," without any attempt to distinguish their real significance or differentiate among them.

It must be evident that after a railway injury, as after injuries occurring under any other circumstances, three classes of conditions may arise: (1) All sorts of injuries of a surgical character; (2) traumatic hysteria; and (3) traumatic neurasthenia. Various combinations of these three may result, and they are commonly found in association. Surgical conditions and neurasthenia or hysteroneurasthenia may be present, or neurasthenia and hysteria may alone follow concussions, injuries, or frights attending accidents, especially upon the railways. It should be clearly recognized that the nervous disturbances marking neurasthenia and hysteria are likely to develop in proportion to the predisposing tendency in the individual, and also in proportion to the amount of mental shock attending the accident. In railway accidents the element of fright reaches its highest development, and consequently there is a preponderance of neurasthenia and hysteria, or their combinations, in persons the victims of such accidents.

In the consideration of a case in which physical conditions and nervous symptoms have originated from injury, it is necessary to look at it first as a surgical case, and secondly to consider it as a nervous case. Cranial fracture, cerebral hemorrhage, focal epilepsy, or traumatic insanity may follow injuries to the head. Dislocations and fractures of the spine, lacerations and hemorrhages of the cord, myelitis and meningitis, muscular strains, and ligamentous ruptures may follow blows and injuries to the back, either directly or indirectly applied. Concussion, if sufficiently severe, even without apparent local physical injury, may induce hemorrhages in the meninges or in the cerebrospinal apparatus. Accidents producing such injuries may at the same time so disturb the nervous equilibrium that neurasthenia is developed or hysteria is provoked. The neurasthenia of traumatism, or of fright associated with the possibilities of traumatism, is exactly the same as neurasthenia arising from any other source. Hysteria associated with traumatism, or conditions associated with traumatism, is exactly the same as hysteria occurring from other causes. The combinations of organic with nervous diseases of a character not yet associated with known organic changes must be deciphered on distinct lines. The surgical features have their own prognosis; the nervous disorders have their proper outlook, and they are not necessarily related.

Cases of this character may be considerably complicated by *litigation.* In exceptional instances there is dishonest and outright simulation. More frequently the anticipation of legal proceedings, the numerous special examinations, the suggestions arising from attorneys and physicians, and the very natural tendency to exaggeration serve to highly accentuate the subjective side of the clinical picture. Corporations and their legal and medical officers usually look upon all such claimants as dishonest, and by their bearing, if not by their words, antagonize and aggravate the patients who come to take an almost morbid, spiteful pleasure in cultivating their aches and bodily and mental distress. They see damages in every symptom, and the hopeful expectation of physical recovery that is so potent for good is completely destroyed. It not infrequently results that, upon the completion of litigation and the cessation of irritation and introspection, prompt improvement takes place. From a medical standpoint, it is always better that an immediate legal settlement be made.

The profession should recognize that traumatic neurasthenia and traumatic hysteria are serious and disabling conditions. Every case must be specialized and the amount of disability and the probability of its duration must be estimated from all the facts.

CHAPTER X.

TICS.

Tic, Maladie des tics, Mimic Spasm, Habit Spasm.—Recent French writers, following Tourette, make a sharp distinction between a tic and other varieties of spasm. After them a tic is a spasm which is identical with movements of volitional intent and contains, therefore, a psychic element which may be subconscious. It is a psychoneurosis. In facial tic they call attention to the winking of the eye, exactly like that which excludes a flying particle of dirt; to the movements about the mouth and nose, identical with those produced by sensations of taste or smell, and to the occasional functional association with these of swallowing efforts, laryngeal motions, the production of sucking or smacking sounds, of grunting, and of articulate words, sometimes of an indecent character (coprolalia). A facial appearance that is expressive of some emotion, as of grief, pain, fright, or joy, may be repeated by the tic. Some cases, becoming more wide-spread, involve the neck and upper extremity so that attitudes and gestures are produced in conformity with the underlying mental idea. These tics, of which blepharospasm is a type and coprolalia the extreme development, are more or less under the control of the patient, who can, by an effort of will, do considerable to repress them. After such repression "tiquers" are likely to feel more or less vague discomfort and often yield to a regular spasmodic debauch, which seems to give them a feeling of relief. During sleep this spasm completely subsides. Such patients frequently present a most marked neurotic heredity, and sometimes other neuroses, as writers' cramp and hysteria, or mental and moral obliquities are present.

Ordinarily, a tic is rapidly executed, and may be repeated with great rapidity from two to scores of times, when a lull occurs for a few minutes, or perhaps an hour of quietude may intervene. Any excitement or embarrassment promptly recalls and intensifies the morbid motions. On the other hand, any decided interest fixing the patient's attention interrupts the twitchings. The spasm has a tendency to invade neighboring muscles of associated function, and frequently becomes bilateral, but is seldom symmetrical, excepting those tics more or less limited to the face, especially to the nose and mouth. While, ordinarily, the spasms are purely clonic, they may, in a part or the whole of their distribution in long-standing cases, present tonic features of greater or less duration. In some instances the eyelids are so firmly closed that the pressure upon the eyeball is painful. Rigidity in the lips is complained of at times as a feeling of stiffness, and the action of the zygomatics and buccinator may keep the angle of the mouth persistently retracted and elevated. The neck may be rigidly held in a given position. A somewhat similar spasm is often noted after Bell's palsy. It is, however, always unilateral, the affected muscles are distinctly contractured (see page 121), and it does not so distinctly imply purpose.

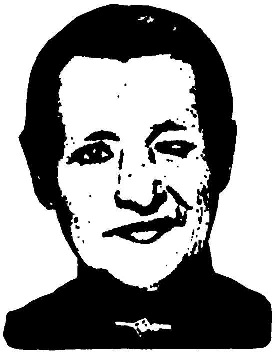

Fig. 266. Facial tic.

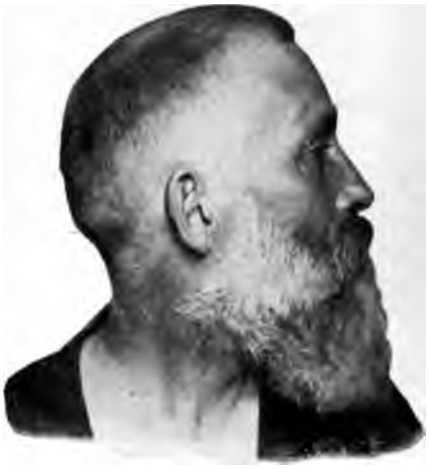

Fig. 267.—Mental torticollis.

Etiology.—Youth is the preferred *age* for the development of tics, but adult life is not spared. An appreciation of the mental substratum of tics enables one to comprehend something of their genesis and intractability. There is no doubt that many cases of blepharospasm originate in some *irritation* of the ocular apparatus that forces the attention of the patient into a groove leading to a mental and motor habit. These tics have for long been well called habit spasms. The term habit chorea, also applied to them, is misleading, though they may be grafted upon a chorea as a sequel by suggestion. In the same way a protracted grief, chagrin, or ecstatic pleasure may, in one neurotically predisposed, furnish the subconscious factor for an expressional tic, which reproduces exactly the facies of the underlying idea, perhaps made grotesque by its unilateral distribution. The thread can sometimes be followed by covering the sound side of the face and trying to interpret the emotion expressed by the tic. Sniffing, swallowing, and phonation are merely the manifestations of functional association, and coprolalia is but the vocalization of the imperative concept that may be otherwise subconscious. A facial spasm is sometimes associated with neuralgia of the fifth nerve, and bears the distinctive and classical name of *tic douloureux;* the spasm, however, is secondary to the pain and expressive of it. It is a grimace, but may persist after pain ceases as a true tic. A tic in the limited sense is not painful.

Varieties.—Aside from the habit movements of idiocy and dementia, which constitute reversionary or degenerate tics, we may enumerate blepharospasm, facial spasm, spasmodic torticollis, mental torticollis, the latah of India, the myriachit of Siberia, the jumpers of New England and Canada.[1]

In mental torticollis[2] some deviation of the head is customary and is spasmodically maintained. Ordinarily, it ceases when the patient lies down or it can be controlled by a slight amount of manual pressure upon the head or face.

Jumpers and the subjects of latah and myriachit execute any one of several commands or suggestions impulsively, often violently, and frequently against their apparent will. Thus, upon command, they may strike, jump, or unclothe themselves.

Treatment.—In all cases of *facial tic*, after general measures, we are to look for and correct any peripheral irritation that can be associated with the seventh nerve. This is particularly the case in the distribution of the sensory portion of the fifth, but irritation arising even at a distance, as in the intestines or pelvis, may maintain the tic, and when corrected the tic may subside. Pressure upon certain points in the distribution of the fifth, first described by Graefe, often checks the tic. The most usual one is at the supraorbital notch. In a general way they correspond to the tender points of Valleix and the maxima of Head. We should always and repeatedly search for them carefully, going over the neck, shoulders, and upper chest, as they are sometimes distantly located. By exerting pressure upon such a part, the tic seems to be reflexly inhibited, and the habit is arrested, at least temporarily. By repeating the arrest frequently and for long periods, the habit may be fully broken. In the same way the use of faradic electricity will occasionally render service. For this purpose the muscles engaged in the tic should be thrown into powerful tonic contractions for a number of minutes, from five to fifteen. One pole taken in the hand and the other placed over the stylomastoid foramen, or on the particular facial branch indicated, makes a suitable arrangement. The patient may advisably keep a battery at hand and use it as often as the tic becomes marked. It for the time being teaches the muscle a steady contraction and perhaps favorably affects the mental state by the peripherally induced suggestion. At any rate such application is often followed by temporary marked relief, and, in fortunate cases, by practical cure. A sugar pill, to be taken with great exactness every thirty minutes, has served a good purpose by constantly reminding the patient of the physician's suggestions, and recalling his attention to voluntary self-control.

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[1] Jos. Collins, "Med. News," Dec. 11, 1897.

[2] Bompairs, "Thèse de Paris," 1894, and Brissaud, "Leçons," 1895.

Finally, nerve-stretching may be employed. If thoroughly done, it induces paralysis for a longer or shorter time, but, unfortunately, a restoration of motor function usually is marked by the reappearance of the tic, for which the irritation in the healing nerve-trunk may be responsible. A tendency to relapse in these cases is marked. The deeper the mental tare and the more pronounced the neurotic background, the less are they manageable. It is not impossible that suggestion, by reaching the subconscious element, may sometimes yield favorable results. All varieties of retention dressings and appliances are at first apparently helpful, but shortly become irksome, unbearable, and harmful.

In *mental torticollis,* Feindal [1] has reported good results by massage and exercises, the purpose of which was to build up the patient's self-control and mental stamina. With these he associated encouragement (suggestion) and general tonic measures. The exercise treatment embraces two varieties of motor effort. First, the patient practises voluntarily controlling the spasmodic muscles by keeping them at rest. Second, the affected muscles are alternately contracted and relaxed methodically a dozen times. These exercises should be repeated hourly. It is only by attacking the mental element, the obsession, that the psychoneurosis can be managed and the various suggested measures are a means to that end.

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[1] "Nouv. Icon. de la Salpêt.," Dec., 1897.

PART VIII.

SYMPTOMATIC DISORDERS.

THERE are a number of symptomatic disorders very commonly met with in nervous maladies and essentially nervous in character that nearly reach the dignity of diseases. Headache, vertigo, insomnia, and neuralgia are the most important. Headache and vertigo have been specifically emphasized whenever they had a special relation to diseases of the nervous apparatus, and can be easily followed from the index. Neuralgia and sleep disorders require further study.

CHAPTER I.

NEURALGIA.

NEURALGIA (nerve pain) is always a symptom. Difficulty arises from the fact that intense neuralgic affections have often been confused with their most prominent painful symptoms. Neuritis and neuralgia have been differentiated only in recent years. Even now there are many who fail to discriminate between trifacial, brachial, and sciatic neuritides, and neuralgias of similar location. It also appears proven that an irritation at first producing neuralgia may, by its persistence, set up a neuritis, and it is certainly clear that neuralgia is one of the symptoms of neuritis. The distinction, therefore, is not always easy. Neuralgia may be an expression of disturbance acting (1) locally, or (2) systemically, or (3) in both ways at once. Neuralgias may be visceral or peripheral, and many visceral diseases have their corresponding peripheral or somatic neuralgias. In this relation the reader is referred to the section on Pain and the Referred Pains of Visceral Disorders as outlined by Head (p. 56, *et seq*). Many neuralgias—such as angina pectoris in cardiac disease, intercostal neuralgia in gastrohepatic disease, testicular neuralgia in kidney disease, pleurodynia, gastrodynia, and coccygodynia —are very clearly symptomatic and secondary. There are certain conditions favoring the appearance of neuralgia, and there are a few localized neuralgias that require individual consideration.

Conditions Favoring Neuralgias.—Intense neuralgic conditions are rare in early life and in old age, but when they appear after sixty, they usually have an irremediable organic basis and are correspondingly intractable. Women suffer in this way more than men. In some instances there is a marked hereditary tendency, and, as a rule, the "nervous" and neurotic, the possessors of unstable nerve-cells, are most prone to neuralgic conditions. This shows out strongly in the clinical histories of hysteria and neurasthenia. Arthritism in its broadest sense is a congener.

The inciting causes of disturbance marked by neuralgic features are very numerous. Any impairment of health, any debilitating influence, any continuous fatigue, anemia, emotion, concussion, may be symptomatized by neuralgia. Exposure to cold, peripheral local irritations, and any cause of neuritis, as well as every form of neuritis, may set up neuralgic pains. Nearly all toxic influences may induce neuralgia. Those toxic causes which are so active in the etiology of neuritis may induce neuralgic pain. Lead, alcohol, diabetes, auto-intoxication, malaria, and acute infections may all have neuralgic symptoms. The root pain of tabes, the peripheral pains of cortical brain disease, and the nerve pains of neuromata are easily understood.

Characters of Neuralgic Pains.—Neuralgic pains are commonly unilateral, and confined to the distribution of a part or the whole of a single nerve, or of several nerves. The pain is inconstant and usually paroxysmal, with complete freedom in the intervals, or with a dull ache between the severe pains. They are usually described as darting, stabbing, tearing, ripping, lightning-like, as if shot, and by other words or phrases expressing their sudden, instantaneous character. They may be repeated rapidly during a few minutes, or occur singly at longer intervals. The pain is located deeply in the parts, but often is attended by great superficial hypersensitiveness. At the same time deep pressure may give relief. During the painful attacks, motion of the muscles of the parts, a touch, even a breath of air in severe cases, may renew the stabbing pains. Heat, cold, and alcoholics may intensify or diminish the pain in various cases.

Neuralgic areas during the pains and between the paroxysms usually present nerve tenderness. This is most prominent at certain points where the nerve is superficial, overlies bone, or is inclosed by fascia or other rigid tissue. They correspond to the "maxima" of Head and constitute the "tender points" of Valleix.

In some cases the pain has a tendency to radiate into other branches of the same nerve or into related nerves. In the same way irritation of one branch of a nerve, especially of the trifacial, may induce neuralgia in another division. Neuralgic pains are often very fugaceous, appearing now here, now there, especially when arising from systemic causes.

The associated muscles may act spasmodically in the pain storms. This is rather common in the trifacial form, but it is difficult to tell whether the grimace is volitional or not. In the same way a sharp leg neuralgia may be attended by a drawing up of the limb, and, if the patient is walking, he may suddenly fall.

Very often the parts subject to neuralgia show vasomotor disturbance and trophic changes. The vessels, at first constricted, usually dilate, and flushing follows. Edema, local sweating, erythema, scaliness, loss of hair, blanching of the hair, herpes, and pigmentation may attend upon neuralgia that is symptomatic of a neuritis. Localized hypertrophy, due to the continued congestive features of neuralgia, may be encountered.

Pathology.—The mechanism of neuralgic pains has been a fruitful theme of discussions, into which we need not enter. The following facts indicate the interposition of the spinal apparatus in neuralgic pains: (1) The pain may occupy the areas related to several spinal segments, and not closely follow the distribution of nerves. In shingles, for instance, the herpetic and painful area on the upper trunk is bounded by horizontal planes, and not by the intercostal furrows; (2) irritation of one branch of the nerve may be radiated into another, which it could only reach by way of the nuclear cells; (3) the pain may develop exclusively in another region than the one irritated; (4) division of the posterior nerve-root, as has been done, especially by Abbé and others, or separation of the afferent path anywhere between the cord and lesion, immediately stops the neuralgia; (5) irritation of the proximal stumps of a divided nerve gives rise to pain referred to the periphery to which that nerve is anatomically related. This is seen in amputation neuromata and in anesthesia dolorosa.

By this conception we are able to understand how systemic poisoning, as by alcohol or malaria, may so predispose the spinal ganglia that pain is occasioned by a peripheral disturbance, perhaps insignificant in itself. It also explains the ability of an intense or protracted peripheral irritation to set up localized pain, which may long persist after the irritant condition has subsided.

Varieties of Neuralgic Pains.—An enumeration of the common varieties of neuralgic pains is all that need be attempted. They are classified (1) as to location, and (2) as to cause.

Varieties Depending upon Location.—Trifacial, cervico-occipital, cervicobrachial, brachial, dorso-intercostal, intercostal, lumbo-abdominal, spinal, sacral, coccygeal, sciatic, crural, metatarsalgia, etc. Visceral forms: Pleurodynia, angina pectoris, cardialgia, gastralgia, gastrodynia, hepatalgia, enteralgia, nephralgia, ovaralgia, testicular neuralgia, etc.

Varieties Depending on Cause or Association.—Epileptiform neuralgia, really a neuralgic facial tic; reflex sympathetic neuralgia, one in which the pain appears at a distance from its irritant source; traumatic neuralgias, really traumatic neuritis; occupation neuralgias, a part of occupation or fatigue neuroses; herpetic neuralgias, the neuralgic pains attending zoster; hysterical neuralgias, really stigmata of the neurosis; rheumatic, gouty, diabetic, anemic, and malarial neuralgias, associated with, and often due to, the respective systemic states; syphilitic neuralgia, very rare, and due to the syphilitic cachexia. Pains in syphilis are ordinarily due to neoplastic infiltration of the nerves and other tissues; degeneration neuralgia appears in the aged, and is due to involutional changes in the organism.

Trifacial Neuralgia.—Of all varieties of neuralgia, that occurring in the trifacial is the most important. It often is extremely persistent and intractable. The constant exposure of the fifth pair in the face and nasopharynx to injury and infection of the periphery, the course traversed by the nerve through bony channels and over resisting structures, and its very extensive distribution territory, lay it especially liable to irritating and traumatic conditions. Its association with other cranial nerves sometimes causes it to be reflexly affected, as from the motor oculi and pneumogastric. It is affected about equally often on either side, and very rarely bilaterally. Exceptionally, all three branches are painful, but more commonly the neuralgia is confined to one or two of them.

When the *first branch* is affected, the pain is supraorbital, radiating from the supraorbital foramen over the corresponding side of the brow, or even to the vertex. The eyeball is frequently tender, or may be the seat of neuralgic pains. Tender *points* are usually found at the notch, on the upper lid, and over the lower margin of the nasal bone. When the *second division* is affected, the pain is located over the cheek, between the orbit and the mouth, spreading onto the wing of the nose. The tender *points* are at the lower border of the nasal bone, over the malar prominence, at the infraorbital foramen, on the gum above the canine tooth, and sometimes on the hard palate. In neuralgia of the *third division*, pain traverses the lower jaw and tongue and the corresponding portion of the face, extending, by the auricular branches, to the zygomatic, and even to the parietal region. The tender *points* are over the inferior dental foramen, in the temple, and in the parietal regions.

The pain is usually intense, lancinating, shock-like, and may cause the most excruciating torture. The attacks, if at all severe, usually cause vasomotor and secretory disturbance. Lacrimation, salivation, and mucous discharge from the nose may be encountered. The brow, or lip, or tongue, or the entire side of the face, may be swollen and edematous. The hyperalgesia is often intense, so that wiping the nose or eye, taking liquids into the mouth, and mastication are attended by great suffering, and often provoke a repetition of the neuralgic pains. Herpes possibly only occurs when histological changes in the nerve or its nucleus have taken place.

The neuralgias of the brachial, intercostal, and sciatic nerves are often intense, and present similar tender points and superficial hyperalgesia. The location of both have been described on page 56, *et seq.*

Treatment.—The treatment of a symptom is necessarily the treatment of the underlying disease. Local and constitutional conditions capable of determining neuralgic pains must be carefully sought, and local conditions capable of producing neuralgia at a distance must not be overlooked. In order to specialize the matter we may consider the treatment of trifacial neuralgia in detail. With proper variations the same considerations and measures apply to other neuralgias.

Treatment of Trifacial Neuralgia.—In the treatment of a trifacial neuralgia a careful search for local irritation is first to be made. In many cases pressure upon a given point will check or inhibit the pain. If such an inhibiting point can be found, it is a source of great relief to the patient. If the pain is intense and the hyperalgesia severe, the use of morphin or cocain may be required to make the examination. This should commence at the vertex. The scalp and brow should be carefully palpated, the orbit investigated, the eye examined for refractive errors, local inflammation, and glaucoma. The nasal fossæ, the antra, the nasopharynx, the mouth, and especially the jaws, must be thoroughly scrutinized. It is useless to sacrifice teeth unless a competent dentist finds them diseased. As a rule, a careful dental overhauling is a necessary measure in protracted cases, even where the pain is not located in the dental branches.

The general systemic state is of equal importance. The facial neuralgias of infections, grip, malaria, and eye-strain commonly involve the ophthalmic division. Dental and maxillary disease is most common in the middle branch. Compression of the nerve in the dental canal often causes neuralgia of the third branch. In elderly people who have lost their teeth the resulting greater elevation of the chin stretches the dental branch of the third, and may cause a neuralgia that can be readily relieved by the use of dental plates of proper vertical proportions to prevent the tug upon the affected nerve. Anemias, cachectic states, and conditions of auto-intoxication from the kidneys, stomach, or intestines, must be corrected. Neoplasms in the cranial cavity, or facial fossæ, jaws, and antra, may impinge upon the nerve and set up neuralgic pains.

In the management of these cases it is usually necessary to maintain complete rest. Many cases, otherwise rebellious, improve very rapidly under the Mitchell rest system. Food must usually be taken in a liquid form, and in very severe cases the nasal tube must be employed. Mastication ordinarily provokes the pains. Directed against the pain we have in malarial cases to employ large doses of quinin, or Warburg's tincture, or both, for several days, and follow them with arsenic, iron, and quinin in moderate doses for weeks. The purpose is to cinchonize the patient and maintain a saturation of the blood with quinin. A preparatory calomel purge is of importance. In other cases we have to resort to sedatives, and are usually reduced finally to the use of morphin. The reliable aconitia of Duquesnel, in doses of $\frac{1}{300}$ of a grain, several times a day, is sometimes of great service, and may be increased if well borne. Slight numbness of the lips, tongue, and fingers may be expected, and this amount of action can not safely be exceeded. Cocain, by local hypodermatic administration or anodal diffusion, is not reliable, and often acts badly. Morphin should only be given by the physician or a competent nurse. It is well if the patient can be kept in ignorance of the nature of the drug, owing to the tendency which is especially strong in these neurotic cases to the acquirement of the opium-habit. The use of electricity is more often disappointing than otherwise. The positive pole to the tender area, with three to ten milliamperes uninterrupted current for ten minutes, sometimes allays the pain. The current should commence from zero, slowly increase until felt as "warm," and finally decrease in the same way, without shock. The use of strychnin hypodermatically in large, repeated, and increasing doses, the patient being meanwhile confined to bed and carefully fed, as advocated by Dana, is a promising line of treatment. Commencing with $\frac{1}{30}$ of a grain every four hours, doses of $\frac{1}{5}$ of a grain are sometimes well tolerated. It is well to employ general massage at the same time, and, as the pain subsides, massage and vibrations to the tender area and sensitive points may be added with advantage. As a last resort, operation may be advised, but only when a careful, intelligent, and persistent use of general and special measures has failed, or there is evidence of organic changes in the nerve or nuclei. There are three general varieties of operations upon the trifacial: (1) Those for the purpose of section, exsection, stretching, divulsing, or twisting out the various branches at some point below the floor of the skull; (2) those directed to enucleation of the trifacial ganglion; (3) division of the sensory root of the fifth above the ganglion. Operations upon the ganglion are very difficult and dangerous. Frequently, the eye on the same side has been lost. The operation of Horsley, in which the skull is opened and the sensory root divided beneath the pons, is much less mutilating and disfiguring, but seems to be attended by danger to life. After root divisions the fibers degenerate upward,[1] thereby producing a permanent result. This operation is worthy of more frequent employment, in spite of its difficulty and danger. Spiller and Frazier[2] modify this proceeding by reaching the sensory root above the ganglion but outside the skull, the operation being carried out precisely on the lines of excision of the ganglion. Operations on the branches below the ganglion usually give temporary freedom from pain, but relapses are common in the same or in adjoining branches. (For details the student is referred to surgical works.) Fortunately, with proper systemic and local treatment these severe operations are very rarely demanded. As the slighter ones of neurectomy, etc., occasionally give permanent relief, they should be tried first.

CHAPTER II.

DISORDERS OF SLEEP.

THE disorders and disturbances of sleep, while mainly symptomatic, in some instances reach an important development and almost attain the dignity of a disease. Sleep may be defined as a recurring, necessary state of lessened muscular, mental, and organic activity, attended by comparative unconsciousness of surroundings. No physical or mental function is absolutely abeyant. Respiration, circulation, metabolism, catabolism, muscular movements, and dreams demonstrate the persistence of functional activities. Entirely dreamless sleep probably does not occur. Sleep, therefore, is a composite which may be variously disintegrated. Sleep-walking, talking in the sleep, nightmare, night-terrors, and nocturnal enuresis, may be considered as localized or partial sleeplessness. In somnambulism the motor apparatus is awake, as it is in night-terrors. In enuresis we have a somnambulism of the lumbar cord, to adopt a term from the French. Of the physiology of sleep, though it is more necessary to life than food, we know comparatively little.

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[1] L. F. Barker, "Jour. Am. Med. Association," May 5, 1900.

[2] "Phila. Med. Jour.," Dec. 14, 1901, and *ibid.*, Oct. 25, 1902.

Physical Features of Sleep.—In sleep there is *muscular relaxation*. The lids are lowered over the upturned eyeballs; the expression is one of repose. *Respiration* is slower and less deep. Mosso states that the amount of air inspired by a normal man during sleep is one-seventh of that used during similar periods of quiet wakefulness. Breathing is distinctly thoracic in character, the diaphragm acting but slightly; inspiration is more prolonged and the respiratory pause is absent. There is a decrease in carbonic acid elimination and an increase in the absorption of oxygen. The *circulation* presents important modifications. The pulse is less rapid. The superficies of the body has an increased vascularity and is often reddened; there is lowered arterial pressure and a smaller central circulation.[1] The brain is comparatively anemic. The arterial changes take place in a precise way, increasing with some rapidity during the first hour, maintaining a maximum for an hour or two, and then gradually decreasing to the waking moment. Such curves have been shown by the plethysmograph and correspond fairly well to those indicating the depth of sleep as demonstrated by Kohlschütter and others. In accordance with universal experience, the first few hours of sleep are, therefore, most profound, refreshing, and valuable. The thorax and limbs actually increase in size during sleep, owing to the circulatory conditions; and we readily comprehend the *increased activity of the skin*, the tendency to *night-sweats*, and the ease with which one is chilled during sleep. Lombard, Rosenbach, and others have observed that the muscle *reflexes* are exaggerated just before

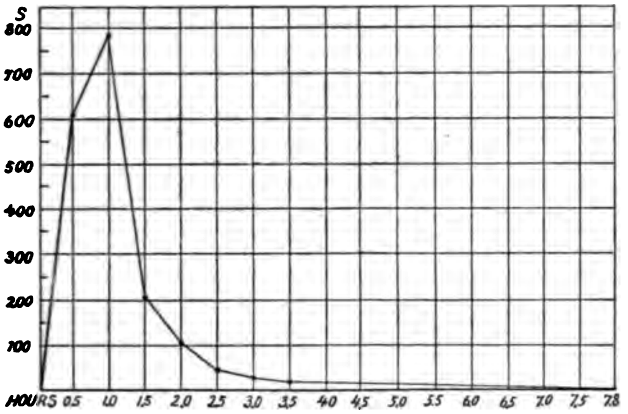

Fig. 268.—Curve illustrating strength of auditory stimulus (falling ball) necessary to waken a sleeping person. The hours are marked below, and the tests were made at half-hourly intervals. The curve shows that the height from which the ball must be dropped reaches its maximum at the end of the first hour (Kohlschütter).

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[1] Howell, "Jour. of Experimental Med.," 1897.

and during the early moments of sleep, but gradually subside, and the knee-jerk may even disappear. Slight disturbances tending to awaken the individual are attended by a corresponding increase in the reflex activity and the central circulation. We are all familiar with sudden startings when dropping off to sleep—a manifestation of the increased reflex excitability, constituting one of the epiphenomena of sleep. The *pupils* are contracted in proportion to the profoundness of the sleep.

Requirements for Sleep.—Individuals vary greatly in the amount of sleep required, and at different ages there are different needs. While some few adults find four or five hours sufficient, the great majority demand eight or ten. There are also temperamental differences and many variations, the result of habit and circumstances. Crichton Brown says that at four years twelve hours' sleep are needed; at fourteen years, ten hours; at seventeen, nine and one-half hours; then seven or eight hours during adult life. In old age continuous sleep is rare, and the requirement is less; but frequent naps and dozing, by day as well as at night, maintain a fair average. Infants may sleep most of the twenty-four hours. More sleep is required in cold than in temperate or warm countries.

Conditions Favoring Sleep.—A cool, dark, quiet, well-ventilated room, a comfortable bed, and adequate, not excessive, covering, are conducive to sleep. A preparatory period of sleepiness is natural, and, in cases of insomnia, must be cultivated. Active emotions, mental worries, intense thought, cold extremities, or a chilled skin defeat the rearrangement of the circulation, on which so much depends. No physical function is so readily disturbed as sleep. If a person is awakened at an unusual hour several nights in succession, he tends to establish a habit of awakening at that hour. Habit is all-powerful, both for good and evil, in this matter. Regular hours for retiring and awaking are most important. The use of hypnotics to put patients to sleep regularly for a few nights do good mainly by reëstablishing the natural initiative.

Disturbances of sleep are (1) those presenting a deficiency, and (2) those marked by a morbid increase of sleep.

WAKEFUL DISORDERS OF SLEEP.

Insomnia.—Insomnia is a symptomatic condition, marked by more or less inability to sleep the individual's usual required length of time.

Etiology.—The causes of sleeplessness are numerous. Practically, every deviation from health is marked by disturbance of sleep; but in many instances, once a bad sleep habit is established, it tends to persist, and may be the chief complaint of the patient. Many persons are hereditarily poor sleepers. In such instances every trivial sound or unusual circumstance—a light, an odor, a jar, or even the discontinuance of a customary noise or light—may arouse them. Hunger, over-feeding, indigestion, constipation, intestinal worms, lithemia, uremia, various drugs, tea, coffee, tobacco, alcoholism, fevers of all sorts, malaria, syphilis, lead-poisoning, cerebral hyperemia or anemia, as from cardiac disease or diathetic states, psychic disturbances, discomfort, pain, neurasthenia, grief, worry, old age, mental preoccupation, and intense study are among the principal inciting causes of sleeplessness.

Symptoms.—Some patients readily fall asleep, but shortly awake, and are then sleepless the balance of the night, or merely secure fitful periods of sleep. Others spend several hours getting to sleep, and may then rest fairly well. Still others complain of broken sleep, the night being passed in intervals of sleep and wakefulness, which may be quite uniform in a given instance. As a rule, patients troubled with insomnia are disposed to exaggerate the amount of sleeplessness, and it is a common experience to find such patients sleeping soundly at times, or even for much of the night, if they are put under watch. Much loss of sleep manifests itself in a haggard, weary air, and in lessened muscular and mental force. Appetite, digestion, energy, courage, and good nature are diminished. The patient loses weight, and, in cases of absolute sleeplessness, the loss may equal that due to deprivation of food. The eyes lose their clearness and look dull, and the sclerotic may be congested. The tongue is coated, constipation may be present, and the entire organism is deranged.

Treatment.—The treatment of the symptom insomnia implies the management of the basic condition of which it is a manifestation. All hereditary, digestive, toxic, circulatory, nervous, and reflex causes must be systematically investigated. The examination of a patient complaining of insomnia omits nothing. Very frequently, modes of living, and especially bad sleep-habits, must be corrected before any improvement is secured. The physical state often requires to be thoroughly well reëstablished by baths, proper diet, exercises, and good hygiene. Attention at once falls upon the conditions favoring sleep, as previously outlined. The patient must be instructed in the importance of these minutiæ, and not expect to secure complete relief by a few doses of medicine. As adjuvants, a warm bath, taken quietly at bedtime, not followed by stimulating frictions, is conducive to sleep. A cold pack or an alcohol rub acts well in some cases. The stomach should not be empty. A glass of hot milk, or milk and water, or malted milk, or hot lemonade, or even of hot water, acts beneficially in decongesting the head in cases not anemic. With the hot bath it favors the dilatation of the cutaneous vessels, and establishes the circulatory conditions found in natural sleep. In the same way a pint of beer not only starts the abdominal organs, but flushes the skin. Large doses of whisky are never advisable, although sometimes efficient for a night or two. Anemic cases require cardiac stimulants and blood-makers.

Any drug that sufficiently masters the organism to produce sleep is a dangerous remedy, and should be used with circumspection, and only as a last resort. Of all the hypnotics, chloral alone is uniformly reliable. In cases attended with much nervousness it is decidedly assisted by the addition of sodium bromid in equal amount. The administration of hypnotics should be done with a definite purpose. It is important to exhibit whichever one is selected at such time that its systemic effect may be operative when the sleeplessness is due. In cases experiencing difficulty in getting to sleep chloral may be given thirty minutes before retiring. Cases awakening at one or two o'clock may be given trional, in dry powder, at bedtime, as its action is liable to be delayed for a few hours. A sufficient dose should be used to have a decided effect, repeated several nights, if its action meets the requirements, and then discontinued. In some instances five-grain doses every two hours after mid-day act much better than a large dose at bedtime. If the underlying cause has been corrected, the proper routine will thereby be reëstablished in a few nights. Finally, some cases only yield after a complete change of scene. An ocean or lake voyage is especially valuable, as it is devoid of exhausting excitement and sight-seeing.

Somnambulism.—In sleep-walking the individual acts his part of a dream. The motor apparatus is awake and responsive to the mind. It is sleep with motor automatic activity, and presents a peculiar increase of the subjective powers of the affected person. There is often great keenness of touch and analgesia combined. The special senses may be active or not, but the patient only takes cognizance of those things which pertain to the dream-story. Usually, the pupils do not respond to light, and the face has a blank, apathetic appearance. The eyes may be open or closed. The sleep-walker has no subsequent waking recollection of his somnambulistic acts; but these may be revived or repeated in a subsequent attack. Talking in the sleep is a minor degree of somnambulism, and the state of double-consciousness may be considered as its largest development.

Somnambulism may ordinarily be considered as a neurotic stigma, and is commonly encountered in those of a neuropathic heredity. Puberty is the ordinary age for its appearance, and both sexes are then about equally affected. Later in life there are more female than male cases. The individual attack is often traceable to some mental precedent condition. Even the suggestion of sleep-walking, in the discussion of the subject, has led to its appearance. In other cases the patient carries out in sleep the line of action on which he had been intent before retiring. In most cases presenting repeated attacks there is a similarity of action in all of them, or one attack may continue the action of its predecessor.

The treatment of the condition should be broad enough to cover the neuropathic make-up of the patient, and is most successful in proportion as it is directed to the mental element in the disturbance. If the patient takes with him to bed a firm intention not to walk, it is often sufficient to inhibit the attack. In nervous subjects of impaired self-control a vigorous suggestion, that will be operative during sleep, must be implanted. This, in children, may be accomplished by a system of rewards or deprivations, by a cold spinal douche at bedtime, associated with the suggestion that it is to prevent walking during the night, or by an emphatic admonition from a respected source. The individual attack may be brought to an end by a dash of cold water, or a sharp puff of breath in the face, or by firm pressure over the supraorbital foramina. So rude a shock may be curative, but in highly nervous children is to be used with caution. In adult cases, with frequently repeated attacks, any plan of treatment is likely to fail, and in some instances nightly precautions are necessary to guard against accidents.

Dreams.—Dreaming is perfectly physiological. As already stated, it is probable that dreamless sleep does not occur. Only those dreams, however, are remembered that are particularly vivid or occur during light sleep, the ordinary condition after the third hour. In numerous experiments it has been found that sleepers, suddenly and unexpectedly aroused, can always give an account of the interrupted dream. Dreams, however, have a considerable medical importance. In lowered nervous or physical states the sleep is not profound, and the patient complains of dreaming constantly. The nature of the dreams has a general relation to the physical and mental status. Only the well have contented, happy dreams. Neurasthenics, hysterics, and melancholiacs are tormented by troubled dreams of an unhappy, depressing character. Neurasthenics and hysterics are prone to have some particular, formulated dream that occurs several times in a night or on different nights. The influence of a terrifying dream in hysteria may equal a severe mental shock in the waking moments, and may be the basis of a hysterical, fixed idea, leading to paralysis, anesthesia, contracture, or assertion of attempts upon chastity (Janet).

In some nervous conditions the dream state is projected into the waking state for a few moments, or for a longer time. In hysteria the features of the dream may then persist for an hour or more, constituting a delirious accident. It is a sort of somnambulism, but with greater mental and physical activity. Or the dream may evoke a convulsive crisis. Of the same nature, perhaps, is *sleep-drunkenness*, or *somnolentia*, in which a person, on being suddenly awakened from deep sleep, is apparently maniacal, and may do acts of violence.

In *pavor nocturnus*, commonly called night-terrors, and occurring only in children, the little patient awakes in vague, wild alarm one or two hours after going to sleep. The child screams in terror, clings to its mother, but apparently fails to recognize any one, and can not be quieted or reassured. After a few minutes or more the excitement spontaneously subsides and the child returns to sleep without recollection of the attack in the morning. During the frenzied terror the patient may run from the room, or climb upon the furniture, in a wild effort to escape something. Often the cries imply a fear of being caught by some one or by wild animals.

True pavor nocturnus is of somewhat serious import, as it indicates an unstable nervous constitution, and is a frequent item in the early history of epileptics. Some writers consider it a true neurosis, and, at least, it is a stigma of degeneracy. It is often apparently induced by respiratory difficulty dependent upon faucial adenoids, bronchitis, laryngitis, weak heart, or general asthenia. In mild form it may only suggest *nightmare* or *incubus*, which is a vivid dream, usually traceable to some physical condition or previous terrifying experience. Indigestion, bad ventilation, or mental shocks, fright, worry, etc., give rise to nightmares attended by a feeling of a great weight on the chest, suffocation or falling. Ordinarily, at such moments the patient suspends respiration, or makes distressed inspiratory noises and awakens with a start.

Nocturnal enuresis is a common disorder in weakly and neurotic children. Neurasthenics, hysterics, epileptics, and the entire range of neuropaths give abundant testimony on the subject of bed-wetting. In certain instances it undoubtedly is an automatic action, a partial somnambulism. The patient dreams he is passing water, and voids his urine accordingly. The same mechanism may operate in the more frequent form. Parents report that when this occurs under observation the bladder acts forcibly, and the stream is propelled with vigor. It is not a mere sphincteric relaxation, as is sometimes alleged. Such children, if taken up at night and made to urinate, may repeat the act almost immediately upon being put back to bed, perhaps through suggestion. It is probable that slight distention of the bladder, or the presence of a drop or two of urine in the vesical end of the urethra, gives rise to sensations which set up a chain of ideas eventually leading to the urinary act. It is definitely proven that various external stimuli—as sounds, voices, lights, etc.—may be partially apprehended by the sleeper and woven into his dreams, materially modifying their trend. Internal sensations are no less liable to act in the same manner, and are generally accredited of doing so in nightmare.

The management of nocturnal enuresis demands attention to several factors: First, the general condition of health; second, the reduction of nervous excitability; third, the prevention of a large vesical accumulation; fourth,—and perhaps the most important of all, as in somnambulism,—the establishment of a definite idea of self-control. It is well to withhold liquids toward the evening, and to have the child thoroughly evacuate the bladder at bedtime. Elevation of the foot of the bed by several inches, to gravitate the urine from the neck of the bladder, has been strongly recommended by Mendelssohn. Belladonna reduces vesical irritability, and the urine should be rendered unirritating. Local disturbance in bladder and urethra must be removed and worms expelled. Finally, a strong mental impression is of the greatest value. For instance, Prendergast [1] cured seventy-five out of eighty cases in a boys' orphanage within a short period of time by giving them a cold spinal douche, followed by a quick rubbing-down just before getting into bed. Nothing else whatever was done, and the five remaining cases were much improved. In some cases the first douche had the necessary effect, and the mind remained on guard.

SOMNOLENT DISORDERS OF SLEEP.

The second group of sleep disorders comprises those marked by somnolence, or by an irresistible tendency to sleep.

Narcolepsy is a condition in which the patient repeatedly goes to sleep during the day. The tendency is irresistible, and the morbid sleep may last a few minutes or several hours. It furnishes one of the accidents of hysteria, and may be dependent upon drugs, brain disease, gout, obesity, uremia, diabetes, and syphilis. Some neurotic families present a number of instances of narcolepsy, associated or alternating with severe neuroses, mainly hysteria and epilepsy. It may be an epileptic equivalent, and many cases also present epilepsy. It should always suggest the possibility of epilepsy or hysteria if defective nutrition and the systemic states mentioned can be excluded. In some instances ocular fatigue, due to refractive errors, seems to be the starting-point for the attacks, which are overcome by appropriate glasses. In some instances the sleep in such attacks is disturbed, and the patient may have vivid dreams and talk in a flighty manner. In other cases, as in epilepsy, the patient is comatose and can not be aroused. In the majority of cases the sleep is deep and no dream recollections are obtainable.

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[1] "New York Med. Jour.," July 11, 1896.

Treatment depends upon deciphering and removing the cause, and then breaking up the habit. For this purpose caffein and nitroglycerin or nitrate of amyl, to control the cerebral circulation, are available.

Sleeping Sickness.—On certain African coasts the natives are affected by a supposedly parasitic disease—(filaria?). Somnolence appears at intervals and gradually increases, until the patient sleeps steadily, at first arousing sufficiently to take food, and finally not awaking under any stimulus. In two cases carefully observed by Mott,[1] a chronic diffuse leptomeningitis and encephalomyelitis were found, but no infectious organism. Death usually results from inanition in four or five months, but patients have lived for two or three years. Treatment seems unavailing.

Trance, catalepsy, and ecstacy are hysterical accidents—fractional hysterical convulsions extended into status. They are described, with their treatment, in the discussion of Hysteria.

SLEEP PALSIES.

Persons with disordered systemic conditions—as in anemia, gout, diabetes, women at the menopause, excessive users of tobacco, etc.—may awake with benumbed extremities. The hands and feet are most affected, giving origin to the term *acroparesthesia*. The sensations are described as pricking and numbness, as being "asleep," and some cramping and decided pain may occur. A loss of power is noted if the condition is marked. The disturbance lasts a few minutes or an hour, and is usually relieved by friction and exercising the members. Less frequently one limb only is affected, or both limbs on one side. This affection may occur nightly for months and years, and appear during the day also if the patient fall asleep. Its treatment is that of the underlying state.

In sleep, especially the profound sleep of drunkenness or the coma of anesthesia, pressure on a nerve may set up a neuritic palsy. This is particularly common in the musculospiral, but in operations on the perineum and pelvic contents the lithotomy position is capable of injuriously stretching the sciatic. The brachial plexus may be injured by elevating the arm too forcibly in operation on the breast, or by the anesthetizer. These are hardly to be called sleep palsies, but are definite accidents occurring during sleep.

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[1] "Br. Med. Jour.," Dec. 16, 1899.

Anemic neurasthenic patients sometimes have difficulty in opening the eyes in the morning, but if the lids are once raised by the finger, they tend to remain open. The symptom is called *sleep ptosis*, is usually transient, and commonly passes away with the myasthenia of which it is part. This, or a similar condition, occurs as a part of myasthenia gravis or pseudobulbar paralysis.

HYPNOTISM.

Hypnotism is an artificial, morbid, sleep-like condition produced in susceptible persons by various methods. In it the complex of sleep is more or less disaggregated. Attention within a limited field and automatic motor activity may be retained, but mental initiative, volition, and judgment are much impaired. The acts and circumstances of well-marked hypnotic conditions can not be recalled by the subject unless the hypnosis is revived. It is, therefore, a state of subconsciousness or double consciousness. Different experimenters find varying proportions of susceptible individuals. The sexes are about equally hypnotizable. Those of mediocre self-consciousness, those accustomed to unquestioningly obey,—hence, children and some hysterics,—are the most ready subjects. A great deal depends upon the preparatory conditions and the belief or skepticism of the patient. In those places where daily séances of hypnotism are held and the subject is introduced into an atmosphere of faith and observes the process in others, success in the method is naturally greatest. Experience on the part of the hypnotizer is also an important factor. The whole matter consists of building up an expectant attention. Low-grade idiots, most of the insane, and many hysterics, incapable of exercising continuous attention, are not hypnotizable by ordinary methods. The state is purely subjective to the one operated upon. After a person has once been hypnotized, he subsequently more readily enters hypnosis, and finally the condition is produced almost at a hint. In extreme cases it may be automatically evoked by anything which suggests the method employed in the given case, and the subject becomes the puppet of a trivial sound or flash of light. During hypnosis there is a high degree of suggestibility, so that the subject unquestioningly accepts the dictum of the operator. In somnambulism the special senses and general sensibility may be extremely acute or entirely blunted, responding only along the various lines of the hypnotic field of activity. Autohypnosis is also possible, and is a common trick of East Indian fakirs and dime museum tricksters in this country.

Methods of Hypnotizing.—After the subject is prepared by example or explanation and his coöperation secured, the most common plan, that of Braid, is to cause ocular fatigue by having the patient fix the eyes in unwinking gaze upon a small, bright object held about six inches distant and above the ordinary line of vision. This shortly produces a feeling of fatigue in the ocular muscles, the eyes water, the lids tremble, and the operator, by suggesting sleep, adds to the mental impression of drowsiness. Passes of the hand over the head or down the limbs may be added. Their soothing effect is well known, and soon the eyes close, or the lids are pressed down by the operator, who enjoins the patient to "sleep, sleep soundly, deeper yet, deep sleep," etc. Four or five minutes may be required for this performance, the patient being placed in a reclining or comfortable attitude for sleep. At this point the subject is firmly and authoritatively told that his eyes are closed, that he is asleep, that he can not open his eyes. The operator may start the subject's hands in rotation and command him to continue the movement, asserting that he can not stop until ordered, etc. Other methods contain the same elements of suggestion and are aided by the operator's voice in a similar manner. Thus, continuous gentle pressure on the eyeballs, passes, and stroking, monotonous sounds, fixing the attention, or gazing fixedly at small revolving mirrors (Luys), the patient meanwhile sitting in a darkened room, are among the methods frequently used. A loud sound or a vivid flash of light, after due preparation, may throw the subject at once into deep hypnosis (Charcot). Bernheim fixes the patient's gaze with his own and suggests sleep: "Sleep is approaching, your limbs are feeling warm and gently tingling, your eyelids are heavy, you are going to sleep; you are sleeping, sleep soundly," etc. The subject is aroused by command, by a puff of air in the face, or by stroking the head. If left to himself, he spontaneously emerges from the hypnosis after a variable time, a few minutes or an hour or two.

The hypnotic state varies in intensity from slight torpor to somnambulistic automatism. Only a small percentage of subjects can be put into the deepest phase; and it is rarely required, as, for purposes of therapeutic suggestion, the lighter phases are quite sufficient. Charcot divided the hypnotic state into three phases—the lethargic, the cataleptic, and that of somnambulism. In the first the subject is as if asleep, without changes of respiration, pulse, or temperature. There is usually some analgesia, but the operator's voice is heard and the subject replies to questions. In the cataleptic stage the limbs retain the position given them, or, if set in motion, continue the movement indefinitely, analgesia is complete, and commands are obeyed within the cataleptic range. In somnambulism the subject is automatically responsive in every way to the operator, whom alone he sees, hears, or obeys, unless otherwise instructed. Suggestions are implicitly accepted, regardless of their nonsensical or irrational character, and commands that do not contravene deep-seated moral convictions are unquestioningly executed. Subsequently, the subject has no recollection of his automatic acts or of the conditions under which they were performed, but, rehypnotized, can readily reproduce them.

Uses of Hypnotism.—In spite of the extravagant claims for the therapeutic value of hypnotism, it is as yet of very little service to the physician. Now and again a minor operation may be done under its influence, saving the slight risk of surgical anesthesia, or by its aid a fixed idea may be removed and a delusion dispelled. Under ordinary circumstances the number of susceptibles is so small that its general use is impossible. In hysteria, as elsewhere, it is a two-edged weapon, and the patient may emerge from hypnosis instituted for a minor difficulty and go into severe hysterical convulsions. One delusion may be removed, but another and more serious one of mind-reading or undue influence may be implanted. For obvious reasons, women should never be hypnotized without reliable witnesses, and the public use of hypnotism can only appeal to the morbid. In this connection its power for harm is proven. There is no longer any doubt that its frequent repetition is harmful to the individual. It tends to destroy self-reliance and to make patients imaginative, weak-minded, and neurasthenic. It also has a tendency to bring discredit upon its employer, and in most instances would better be substituted by measures of equal efficiency and less disadvantage. Suggestion, however, is a mighty aid to the physician, and, without producing hypnosis, positive and intelligent assertion can accomplish all that is likely to be done by hypnotism short of the somnambulistic stage. A fair realization of the part suggestion plays in therapeutics is one of the recent achievements of the most progressive medical minds.

MENTAL DISEASES.

BY

FREDERICK PETERSON, M. D.,

EX-PRESIDENT OF THE NEW YORK STATE COMMISSION IN LUNACY; CLINICAL
PROFESSOR OF NEUROLOGY AND PSYCHIATRY, COLUMBIA UNIVERSITY;
CONSULTING ALIENIST, BELLEVUE HOSPITAL; GENERAL CON-
SULTANT TO THE CRAIG COLONY FOR EPILEPTICS, AT
SONYEA, NEW YORK.

MENTAL DISEASES.

CHAPTER I.

INSANITY.

Synonyms.—Psychosis, Psychopathy. *German:* Irrsinn, Irresein, Verrücktheit, Wahnsinn. *French:* Aliénation mentale, Folie.

It is the object of the author to bring together in the following chapters such matter in relation to the definition, classification, etiology, pathology, symptomatology, and treatment of insanity as will be of actual practical value to the medical student and general practitioner.

The seeker after special information and deeper knowledge of the complex subject of morbid psychology must be referred to the many profound works which deal with this exclusively. These chapters are based upon my clinical lectures given at the Manhattan State Hospital for the Insane and at the Randall's Island Asylum for Idiots during the past four or five years, to the students of the College of Physicians and Surgeons of Columbia University, and to the students of the Woman's Medical College of the New York Infirmary. They, therefore, embody only the facts which I believe to be the most serviceable and useful to those who are often practically concerned with the early diagnosis and prognosis of insanity, and who must be the first arbiters as to the course of care and treatment to be pursued.

Definition.—The difficulty of making a rigid definition of insanity is recognized by all who have attempted it. So various are the manifestations of mental aberration, so many the faculties involved, so different the degrees of deviation from the normal, it is no wonder that the expert hesitates and often fails in the effort. The definition, too, must include idiocy, and must exclude certain states of transitory mental disorder, such as the delirium of fevers and of intoxications.

The noted English jurist, Lord Justice Blackburn, once said, while giving evidence before a committee of the House of Commons: "I have read every definition which I could meet with, and never was satisfied with one of them, and have endeavored in vain to make one satisfactory to myself. I verily believe that it is not in human power to do it."

Fortunately, we are not often called upon to give a definition of insanity, and usually we may reply that insanity is a symptom of so many obscure pathological states, and appears in such divers forms that a narrow definition is not possible. However, the practitioner may find himself in the witness-box some day, and it is not uncommon for one of the legal examiners to ask of the witness in a mental case a definition of insanity. If the witness be wise, he will answer as indicated, or he may qualify such answer by offering to quote some one of the definitions given by alienists, such as follow:

A disease of the brain (idiopathic or sympathetic) affecting the integrity of the mind, whether marked by intellectual or emotional disorder.—(Hack Tuke.)

A special disease, a form of alienation characterized by the accidental, unconscious, and more or less permanent disturbance of the reason.—(Régis.)

Morbid derangement, generally chronic, of the supreme cerebral centers,—the gray matter of the cerebral convolutions or the *intellectorium commune*,—giving rise to perverted feeling, defective or erroneous ideation, and discordant conduct, conjointly or separately, and more or less incapacitating the individual for his due social relations.—(Maudsley.)

Insanity is either the inability of the individual to correctly register and reproduce impressions (and conceptions based on these) in sufficient number and intensity to serve as guides to actions in harmony with the individual's age, circumstances, and surroundings, and to limit himself to the registration as subjective realities of impression transmitted by the peripheral organs of sensation, or the failure to properly coördinate such impressions and to thereon frame logical conclusions and actions, these abilities and failures being in every instance considered as excluding the ordinary influences of sleep, trance, somnambulism; the common manifestations of the general neuroses, such as epilepsy, hysteria, and chorea; of febrile delirium, coma, acute intoxications, intense mental preoccupation; and the ordinary immediate effects of nervous shock and injury.—(Spitzka.)

With these few examples before us of the diversity of definition attained by careful students of psychiatry, we may well content ourselves and acknowledge that a satisfactory definition in brief form is scarcely to be devised. The writer has often qualified this by offering the following, which has at least the merit of brevity, if not of perfect adequacy:

Insanity is a manifestation in language or conduct of disease or defect of the brain.

The law assumes to offer certain definitions of insanity, from which, however, those of medicine would tend to differ, in connection with the three chief points where law and psychiatric medicine meet:

1. A criminal is insane if he does an act whose nature and quality he does not know, or if, knowing the nature and quality of his act, he does not know whether it is right or wrong.

2. A testator is insane if his mind, memory, or understanding is unsound.

3. In a lunacy inquisition the subject of the inquiry is insane if he

is incapable of managing himself and his affairs. Such are the divergent tests of insanity in law.

Classification.—What has been said of the difficulty of defining insanity is equally applicable to classification. Not all of the writers of works on psychiatry have deemed it expedient to offer a definition of insanity, but there is scarcely one who has not presented us with an original classification, or one modeled upon, or modified from, that of his favorite authority. It will be impossible as well as useless to attempt to enumerate in these pages one-half of the many classifications which have been made, held for a time, and finally abandoned with the advance of science and the accumulation of new facts in the domains of pathology and psychology. It suffices to say that there are at least forty such classifications which have been made upon etiological, psychological, symptomatological, or pathological grounds. I shall present here, simply as examples for reference, several of the latest and best classifications of the Anglo-American, German, and French schools.

The Statistical Committee of the Medico-psychological Association of Great Britain adopted the following classification for use by the medical superintendents of asylums:

1. Congenital or infantile mental deficiency—
    - *a.* With epilepsy.
    - *b.* Without epilepsy.
2. Epilepsy (acquired).
3. General paralysis of the insane.
4. Mania—recent, chronic, recurrent, *a potu*, puerperal, senile.
5. Melancholia—recent, chronic, recurrent, puerperal, senile.
6. Dementia—primary, secondary, senile, organic,—*i. e.*, from tumors, coarse brain disease.
7. Delusional insanity.
8. Moral insanity.

Maudsley's grouping is as follows:

I. AFFECTIVE OR PATHETIC INSANITY.

1. Maniacal perversion of the affective life (mania without delusion).
2. Melancholic depression without delusion (simple melancholia).
3. Moral alienation proper approaching this, but not reaching the degree of positive insanity in the insane temperament.

II. IDEATIONAL INSANITY.

1. General.
    - *a.* Mania.
    - *b.* Melancholia $\begin{cases}\text{acute.}\\ \text{chronic.}\end{cases}$
2. Partial.
    - *a.* Monomania.
    - *b.* Melancholia.
3. Dementia $\begin{cases}\text{primary.}\\ \text{secondary.}\end{cases}$
4. General paralysis.
5. Imbecility.

Classification of the Congress of Paris (1889):

1. Mania (acute delirium).
2. Melancholia.
3. Periodical insanity (circular insanity, etc.).
4. Progressive systematized insanity.
5. Vesanic dementia.
6. Organic dementia.
7. Paralytic insanity.
8. Neurotic insanity (hypochondria, hysteria, epilepsy, etc.).
9. Toxic insanity.
10. Moral and impulsive insanity.
11. Idiocy.

The following is the classification of Régis:

I. FUNCTIONAL INSANITY.

- Generalized or symptomatic.
  - 1. Mania.
    - Subacute mania (maniacal excitation).
    - Acuta mania (typical mania).
    - Hyperacute mania (acute delirium).
    - Chronic mania.
    - Remittent or intermittent mania.
  - 2. Melancholia.
    - Subacute melancholia (melancholic depression).
    - Acute melancholia (typical melancholia).
    - Hyperacute melancholia (with stupor).
    - Chronic melancholia.
    - Remittent or intermittent melancholia.
  - 3. Insanity of double form
    - continuous.
    - intermittent.
- Partial or essential insanity.
  - Systematized progressive insanity.
    - First stage (hypochondriacal insanity).
    - Second stage (persecutory, religious, erotic, political, etc.).
    - Third stage (ambitious insanity).

II. CONSTITUTIONAL INSANITY.

- Degeneracy of evolution. (Vices of organization.)
  - Disharmony (defect of equilibrium, irregularity, eccentricity)
  - Neurasthenia (fixed ideas, impulsions, aboulias).
  - Phrenasthenias
    - delusional.
    - reasoning.
    - instinctive.
  - Monstrosities (imbecility, idiocy, cretinism, myxedema).
- Degeneracy of involution. (Disorganization.)
  - Dementia (simple dementia).

Krafft-Ebing has drawn up this scheme:

A. MENTAL DISORDERS OF THE DEVELOPED BRAIN.

I. PSYCHONEUROSES.

- 1. Primary curable conditions.
  - Melancholia
    - simplex.
    - attonita.
  - Mania.
    - Maniacal exaltation.
    - Acute mania.
  - Stupor, or curable dementia.
  - Wahnsinn (vesania).

- 2. Secondary incurable states.
  - Secondary monomania (Verrücktheit).
  - Terminal dementia
    - agitated.
    - apathetic.

II. Psychic Degeneracies.

- 1. Reasoning insanity.
- 2. Moral insanity.
- 3. Primary monomania (primäre Verrücktheit—persecutory, erotic, religious, ambitious).
- 4. With imperative conceptions.
- 5. Insanity from constitutional neuroses
  - epileptic.
  - hysterical.
  - hypochondriacal.
- 6. Periodical.

III. Cerebral Diseases with Marked Mental Symptoms.

- 1. Paralytic dementia.
- 2. Cerebral syphilis.
- 3. Chronic alcoholism.
- 4. Senile dementia.
- 5. Acute delirium.

B. ARRESTED CEREBRAL DEVELOPMENT.

- 1. Idiocy.
- 2. Cretinism.

Ziehen has given the most recent, and in many respects the best, classification of this decad :

I. Psychoses without Intellectual Defect.

- A. *Simple psychoses.*
  - 1. Affective psychoses.
    - Mania.
    - Melancholia.
    - Neurasthenia.
  - 2. Intellectual psychoses.
    - Stupidity.
    - Paranoia
      - simple.
      - hallucinatory.
      - ideational (ideenflüchtige).
      - stuporous.
      - incoherent.
    - Imperative conceptions.
- B. *Mingled psychoses.*

II. Psychoses with Intellectual Defect.

- *a.* Congenital weakness (idiocy, imbecility, feeble-mindedness).
- *b.* Acquired weakness, or dementia.
  - 1. Paralytic dementia.
  - 2. Senile dementia.
  - 3. Secondary dementia (after functional psychoses).
  - 4. Secondary dementia (after cerebral lesions, syphilis, etc.).
  - 5. Epileptic dementia.
  - 6. Alcoholic dementia.

Kraepelin, who has done so much of recent years to try to bring the chaos of classification of insanity into some sort of order, and who by his example has so greatly stimulated the clinical study of psychiatry, has adopted the following classification:

I. INFECTIOUS PSYCHOSES.
  - *a.* Fever deliria.
  - *b.* Deliria from infections.
  - *c.* States of exhaustion from infections.

II. PSYCHOSES OF EXHAUSTION.
  - *a.* Collapse delirium.
  - *b.* Acute confusion (amentia).
  - *c.* Chronic nervous exhaustion.

III. INTOXICATIONS.
  1. Acute intoxications.
  2. Chronic intoxications.
     - *a.* Alcoholism (intolerance of alcohol, pathological intoxication, chronic alcoholism and alcoholic deterioration, delirium tremens, Korsakoff's psychosis or chronic delirium, hallucinatory delusional states or alcoholic hallucinosis, acute and subacute forms, and systematized delirium, hallucinatory dementia or alcoholic paranoia, delusions of jealousy, alcoholic pseudoparalysis).
     - *b.* Morphinism.
     - *c.* Cocainism.

IV. THYREOGENOUS INSANITY.
  - *a.* Myxœdematous insanity.
  - *b.* Cretinism.

V. DEMENTIA PRÆCOX.

Hebephrenic, catatonic, and paranoid forms.

VI. PARALYTIC DEMENTIA.

VII. INSANITY WITH CEREBRAL DISEASE (cortical gliosis, diffuse sclerosis, Huntington's chorea, multiple sclerosis, syphilis, tabetic psychoses, arteriosclerotic atrophy, circumscribed lesions—tumors, abscesses, hemorrhages, embolism, atrophies, and traumatic disorders.

VIII. PSYCHOSES OF THE PERIOD OF INVOLUTION.
  - *a.* Melancholia (simple and hypochondriacal forms, depressive delusional states, anxious melancholia).
  - *b.* Presenile persecutory delusional states.
  - *c.* Senile dementia (presbyophrenia, depressions, delirium, paranoid states).

IX. MANIC-DEPRESSIVE INSANITY (manic and delirious states, depressive and stuporous states, and mixed conditions).

X. PARANOIA (of persecution, of grandeur, erotic, and hallucinatory forms, original paranoia, and paranoia of querulants).

XI. EPILEPTIC INSANITY (dementia, periodical moods, delusional states, states of obscuration, stupor, anxious delirium, and dipsomania).

XII. PSYCHOGENOUS NEUROSES.

- *a.* Hysterical insanity (hypochondriacal disorders, change of character, dream states and deliria, moods and states of agitation).
- *b.* Neuroses of fright.
- *c.* Neuroses of anticipation.

XIII. ORIGINAL OR CONSTITUTIONAL CONDITIONS.

- *a.* Nervosity (including congenital neurasthenia).
- *b.* Constitutional depression.
- *c.* Constitutional excitement.
- *d.* Imperative psychoses ("manias" and phobias).
- *e.* Impulsive insanity (kleptomania, etc.).
- *f.* Sexual psychopathia.

XIV. PSYCHOPATHIC PERSONALITIES.

- *a.* The born criminal, and moral insanity.
- *b.* The unstable (including pseudodipsomania and habitual criminals).
- *c.* The morbid liars and frauds.
- *d.* The pseudoquerulants.

XV. STATES OF DEFICIENT DEVELOPMENT.

- *a.* Imbecility.
- *b.* Idiocy.

To any but the expert and special student some of these classifications must, indeed, be mystifying and incomprehensible. They are forbidding to the ordinary student and to the general practitioner, and might well induce him to shun the realms of psychiatry which open before him so uninvitingly and present such obstacles to his progress. And the fact is that they are interesting to the specialist alone because they are as yet quite impracticable from the standpoint of actual utility, as is evidenced by the employment even by the physicians of asylums, who are nothing if not practical alienists, of far simpler schemes of classification in the preparation of statistics for their annual reports and in the histories entered upon their case-books. If the asylum practitioners are compelled for practical purposes to adopt a simple method of classification, how is the novitiate in psychiatric learning to surpass them in the diagnosis and grouping of his cases? Here, for instance, are two of the latest classifications made for the asylums of New York State by the State Commission in Lunacy, assisted by the director of the Pathological Institute and a committee of superintendents of the hospitals (1905).

The first of these tables is one for statistical purposes only, it having been found to be more practical to separate the statistical classification from one devoted entirely to clinico-pathological purposes:

STATISTICAL TABLE OF NEW YORK STATE HOSPITALS.

1. Alcoholic insanity.
2. General paralysis.
3. Senile insanity.
4. Epilepsy with insanity.
5. Imbecility and idiocy with insanity.
6. Other psychoses.
7. Not insane.

CLINICO-PATHOLOGICAL CLASSIFICATION OF NEW YORK STATE HOSPITALS.

1. Brain tumor.
2. Traumatic psychoses.
3. Psychoses accompanying other nervous diseases.
4. Senile psychoses.
5. General paralysis.
6. Alcoholic psychoses (with subdivision into types).
7. Morphinism and cocainism, etc.
8. Infective-exhaustive psychoses (delirious types).
9. Allied disorders.
10. Depression not sufficiently distinguished.
11. Melancholia symptomatic.
12. Depressive hallucinosis.
13. Involution melancholia.
14. Disorders allied to the depressions.
15. Paranoic conditions.
16. Dementia præcox.
17. Allied disorders.
18. Manic-depressive psychoses (first, second, third, fourth, etc., attack).
19. Allied disorders.
20. Constitutional inferiority.
21. Hysterical insanity.
22. Epileptic insanity.
23. Imbecility and idiocy with insanity.
24. Not classified.
25. Not insane.

In the above, the item 9 implies disorders akin in type, but not sufficiently in etiology, to the toxic and infective-exhaustive processes. Item 14 covers disorders allied to the depressions (Nos. 10 to 13). Item 17 contains the disorders akin to paranoic conditions and dementia præcox.

With this direct evidence of the practical necessity of a simple grouping of cases into forms of insanity which are readily distinguished from one another, I have felt that I could not do better in my teaching than to adopt some similar arrangement. It took me a long time, after beginning a residence of several years in a large insane asylum, to crystallize the types, which were at first very confusing, into some sort of systematic division. The student or practitioner unfamiliar with insanity would, upon a visit to a large institution, probably be first struck by the peculiar shapes of heads of some of the patients. He would find, upon inquiry, that the cases with malformed crania were congenital idiots, imbeciles, or feeble-minded, and before much time had elapsed he would unconsciously make in his own mind the two great divisions of the insane into those with *diseased brains* and those with *defective brains*. The former class includes the brains which had developed normally for years and had then fallen a prey to disease; the latter the brains affected by congenital defect or by defect acquired through organic diseases in earliest childhood. In other words, he would separate them first into the *insane proper* and *idiots*. The term *idiocy* comprises three degrees of mental impairment—*profound idiocy*, moderate defect or *imbecility*, and mere weakness of mind, or *feeble-mindedness*.

Now, as the student proceeds to study the physiognomy, conduct, and speech of the cases of insanity proper, he distinguishes, ere long, states of depression (the gloomy visages and unhappy ideas of melancholia) and states of exaltation (the ideomotor excitement of mania). Their histories will tell him whether they are acute, subacute, chronic, or recurrent, and whether they are puerperal, lactational, climacteric, senile, epileptic, hysterical, toxic, etc., in their origin. A very small percentage of cases presents the curious phenomena of alternating phases of mania, lucid intervals, and melancholia, giving the entity a cyclical character, and these rare examples of mental disorder are distinguished as circular insanity.

Epilepsy is found in a considerable proportion of the inmates of asylums,—sometimes conjoined with idiocy, sometimes with dementia, sometimes with symptoms of mania or melancholia, and often presenting peculiar qualities of mental disorder which render the subject of *epileptic insanity* worthy of a separate chapter. A very large group of patients in the asylum would probably impress him as being idiotic or imbecile by their speech and demeanor, and yet he would observe them to have normally shaped heads, and traces of old intelligence might be manifest. By inquiry into the histories of these numerous patients he would find that the mental enfeeblement so plainly apparent had been a sequel to a serious antecedent brain-storm, had followed upon an attack of mania or melancholia, and that the designation of this condition is accordingly *secondary* or *terminal dementia.* The term "dementia" means in psychiatry enfeeblement of the mind. There are rare cases where such enfeeblement develops *ab initio*—*e. g.*, without either of the antecedent psychoses just described, and this condition is entitled *primary dementia.*

Still another considerable group of cases becomes distinct upon further study, a syndrome identified by progressive mental enfeeblement, accompanied by progressively increasing paresis or paralysis of the muscles of speech, of the face, of deglutition, of the limbs, in fact of the whole body,—dementia paralytica, general paresis, or general paralysis of the insane,—with its phases of grandiose ideas, tremor, epileptiform and apoplectiform episodes, exaggerated or lost knee-jerks, and Argyll Robertson pupils.

He would then begin to be puzzled by the fixed delusions of persecution and grandeur, especially by the former, which he would encounter in many patients,—in some rather confused, weak, and transitory; in others systematized into a most elaborate scheme, with considerable logic and intelligence. Some study of these would lead the student to place the less elaborate persecutory ideas among the melancholiacs, alcoholics, etc., the less elaborate grandiose ideas among the chronic maniacs and general paretics, while the wonderfully elaborated delusions of grandeur and persecution of *paranoia* would lead him to recognize under this heading the remarkable mental disorder known to the Germans as "primäre Verrücktheit," to the English as chronic delusional insanity, and once familiar to us by the rather indefinite term of monomania.

I shall leave for discussion under separate headings any further subdivisions that seem to me useful. The outline just given must serve the purpose of a foundation upon which the student will rear such superstructure as his time and inclination may permit. Accordingly, the chapters on special forms of insanity in this book will be simply arranged as follows:

1. Mania. } Manic-depressive insanity.
2. Melancholia. } Manic-depressive insanity.
3. Circular insanity. } Manic-depressive insanity.
4. Epileptic insanity.
5. Dementia (primary and secondary). Dementia præcox.
6. General paresis.
7. Paranoia.
8. Idiocy, imbecility, and feeble-mindedness.

A REVIEW OF RECENT PROBLEMS OF PSYCHIATRY.[1]

Most text-books of psychiatry show plainly an effort at harmonizing one's daily experience with that which one may consider the safe scientific generalization of the age or of the individual. Some writers do it chiefly for didactic purposes, in order to shape the mental attitude of students; others more especially because their experience is continually fermenting in them, and demands an orderly arrangement in order to give them peace and satisfaction. In any such effort we may distinguish those whose tendency goes more in the direction of harmonizing the facts of experience with a "science" which they consider established and having a set form, while others set the facts of experience foremost, and adapt the order of the science to their needs. The large number, of course, of those who practice the specialty are on free empirical ground—persons who are more or less professedly, and perhaps with preference, without system, and get along best without taking a definite stand of their own on general questions, and who, if they be teachers, render the general consensus without any special interest one way or another.

After all, progress is possible without any very broad scientific system. The first need is a knack of observation and of seeing distinctions; and the next, to draw the necessary inferences, to see their most suggestive sides, and to use this material properly. We see, however, that discoveries of the unsystematic are easily used for sweeping generalizations, which are proved or disproved only in the course of time, as general theories and especially many so-called discoveries in therapeutics show. The more the discoverer has a systematized and scrutinized stock of facts and a keen eye for illusions of perception and of reasoning, the more is he likely to put forth his findings in a sound permanent light.

In the history of scientific progress undoubtedly those with a need of setting things in harmony with the safe data have been the chief promoters, and in order to get an idea of the most modern phase of our specialty we propose to analyze the work of a few of these pathfinders for the purpose of seeing before us the plan of some lines of work which have shaped the general trend of interest, and which, as the Germans say, made schools, *i. e.*, were the start for whole sets of new investigations along the newly opened paths. Very striking generally acknowledged transformations in this young specialty of medical science are not so abundant that they may not be reviewed with some ease at this date.

In neurology and psycho-pathology nothing has created so profound a commotion as the discoveries connected with the study of *aphasia*, which were closely followed by that of the other facts of cerebral localization. The most fundamental discovery, from the alienist's point of view, is undoubtedly that of Wernicke, in his demonstration of sensory aphasia. The earlier discovery of motor aphasia had not really pointed so forcibly to the need of an inner analysis of the processes of speech and thought, being too largely like the loss of a tool, and not an intrinsic disorder. In line with the discovery of sensory aphasia, we note, also from Wernicke's school, the great development of our knowledge of mind-blindness, and of apraxia.

In psychiatry proper we see a large amount of valuable work grouped around the problem of *etiology*. The mere psychological description of types of mental derangement being so very complex,—apparently an almost endless task with endless disappointment and disagreement,—the problem of heredity attracted the attention of all through the work of Morel and his followers. It has established facts which are, however, in much need of explanation themselves, and not very productive of further knowledge. Their present influence is largely in pointing to the necessity of a future study of constitution, and to caution in questions of etiology generally, inasmuch as they show the need of considering a special predisposition besides the exciting causes. For the most important result to be expected from it in the future, the question who is entitled to progeny, the facts as well as human society are far from being mature.

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[1] This review, particularly of the work of Kraepelin, Ziehen, and Wernicke, has been prepared by Dr. Adolf Meyer, Director of the Pathological Institute of the New York State Hospitals.

A very stirring turn in psychiatry has come lately from Kraepelin, who took a stand foreshadowed by Kahlbaum, but never demonstrated with such consistency before. With the fifth edition of his text-book, the time had come to break away from the resigned symptomatic attitude. The pride of the clinician demanded disease-entities, and conditions seemed mature enough to claim at least a large number of the types and the cases for definite pathological or at least nosological entities. This was achieved by the consideration of the evolution of the whole life in insane patients, and utilization of a retrospective review of the development in such as had reached terminal stages. Kraepelin was encouraged by such a review, owing to the similarity of the outcome of the disease, to form groups of cases which had been regarded as distinct before, because they represent what seem to be distinct symptom-complexes. To start with, Kraepelin showed that they might be diagnosed to better advantage and with a prognostic forecast from the very start if certain facts were taken into account. His emphasis of the nature of *course and outcome* for the shaping of "disease-entities" has certainly freshened interest in observation remarkably. He was in part pushed to this attitude by the extreme stand of a former assistant of Kahlbaum (Ziehen), who subsequently developed a psychology and psychiatry on the basis of a modernized Herbartianism, and association-psychology. A review of Ziehen's psychiatry by Kraepelin brings the contrast between psychological and clinical psychiatry to a culmination, and the fifth edition of the text-book was the book Kraepelin promised in his review of Ziehen's work ("Zeitschrift für Nervenheilkunde," 1894).

While these two extremes developed, Wernicke had gradually published his "Psychiatry," which stands now before us as the most recent fundamental work, and which deserves very careful analysis by every one who is interested in the development of psychiatric thought, owing to its clinical accuracy and the efforts at explaining mental diseases as disorders of function of the nervous system.

This provisional summary is, of course, not a complete picture of what I consider valuable psychiatric literature. There are many excellent works of a less extreme attitude which probably depict much more satisfactorily the current standing of the specialty, but which are more didactic and not likely to inspire to research. Most of the English and American text-books belong in this category; also many of the French and Italian works, and in German literature especially Schüle, Krafft-Ebing, and Sommer, who depicts the modern trend.

Before entering into the details, we wish to give the reader a brief characterization of the tendencies which we propose to analyze. This may lead to preconceived ideas; but any harm will, we hope, be corrected by the expected effect of the review, that the reader will feel sufficiently interested to go himself to the original works, and a preliminary outline will bring out the issues more rapidly and clearly.

The average work on mental disease cannot quite escape the comment that it is an attempt to give a list of accepted traditional terms of mental disease some justification, in the face of an obviously much larger variety of cases than these terms and standard descriptions can cover. The result is rather unsatisfactory. I am, therefore, not surprised that most of the alienists with whom I come in contact consider it hardly worth their while to keep track of the interpretations given by more than a few text-books which they take as standards.

The types of "psychiatries" which we are about to discuss are not quite free of this charge, since in order to be understood at all, one must use the language and the terminology as it is, and trust that the reader will respect one's definitions, and not give the names and words an arbitrary meaning. These definitions must start from views which one has a right to consider as held in common, and any attempt at inventing too many new names meets a prompt revenge, as the fate of the books of Kahlbaum and Arndt have shown. But we can claim, for all the writers discussed here, a conscientious effort to start in the first place from sets of patients, and to adapt their descriptions and terms to what they see, without any unnecessary regard for traditional classification of words.

This is perhaps least obvious with Ziehen. With him, the system of available terminology is responsible for his divisions to a great extent. In his general pathology the system of his association-psychology also furnishes the backbone for pathological psychology. Given the headings of the chapter in normal psychology, one can construct with ease the subheads of the chapter on pathology.

This form is naturally dry, and does not quite do justice to the readability of the very good observations of fact which are classified in this very systematic manner. The student may be grateful for the beautiful order; but the practitioner does not see the things as the teacher puts them down. What is to him an entity of great precision and value is torn apart here, and he naturally asks, To what advantage? The beautiful order does not dispel his fear that over the details he might fail to see the otherwise familiar and practically useful entity.

When we come to the division on special psychiatry, the description of the actual disease-types, the psychological order is again uppermost, as we shall see later in our analysis.

Kraepelin is diametrically opposed to this in his latest editions. It is true that he, too, sought his salvation in psychology, and classifies the facts in his general pathology somewhat according to the headings of Wundt's psychology. In the former editions he had but few headings of special disease-types, mostly in harmony with the accepted terminology built on a few standard types of disorders which are supposed to be open to psychological classification. Of these, I mention the standards: States of depression (simple melancholia and melancholia with delusions); states of mental obscuration (hypnotism, somnambulism, epileptic and hysterical states; stupor and ecstasis, and acute dementia); states of excitement (melancholia activa, mania, delirium); periodic psychoses (mania, melancholia, circular forms); paranoia; paralytic dementia; states of psychic defect.

In the fifth edition Kraepelin breaks loose from the satisfaction with this symptomatic compromise and proclaims the need of broader nosological principles; medical interests, not a psychological system, shall decide the classification; the medical questions of course, prognosis, and outcome demand the first place: and among these the prognosis is the most important. He divides most of the so-called acute disorders into recoverable forms and psychoses of deterioration, and makes this division an important principle of classification. Superficially very heterogeneous symptom-complexes are thus brought under one head, and broader, not necessarily psychological or etiological, facts are the new guides.

Wernicke is very decided in his antagonism to Kraepelin's sweeping innovation. He considers such an undertaking premature. Probably this is an extreme view. But he has a great point in his favor, that of frank admission that a complete comprehension of all the psychoses is not to be expected to-day, that certain groups of cases, however, can be sufficiently understood and harmonized with our knowledge of neurological and psychological facts. On the other hand, he condemns all attempts to obscure the line of distinction between what is clinically known and what is not known, even if this be done under the appearance of leading to broader concepts. We shall see that he himself does not use the same reserve in his own theories of localization.

A very concise and clear statement of Wernicke's position is contained in his article on the classification of psychoses.[1]

He makes due reference to his predecessor, H. Neumann, who recognized only one "insanity," or as many mental disorders as there are diseased persons, in contrast to the dogmatic Griesinger, who built a psychiatry on psychological ideas with observations borrowed from the literature. Neumann was one of the founders of a conscientious symptomatology. He made a deep impression on Kahlbaum, whose work Wernicke considers the most valuable to this day,[2] although it is evidently encumbered by haziness owing to his desire to do justice to Neumann (especially the idea that all typical insanity shows four stages: melancholia, mania, confusion, and dementia). Kahlbaum's great merit is the distinction between Habitualbild (or Zustandsbild), *i. e.*, the temporary symptom-complex, and the actual underlying disease; and the recognition that most of the so-called forms of insanity are merely pictures of symptom-complexes without sufficient analysis of their nosological bearings. Wernicke next mentions Meynert, who adheres to the current headings of mania, melancholia, secondary insanity, and dementia, and classifies all the rest of the acute psychoses as amentia. He finally mentions Kraepelin's classification as probably the most popular, and claims that notwithstanding Kraepelin's emphatic defense, it is essentially an etiological grouping.

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[1] "Psychiatrische Abhandlungen." Heft 12. Breslau, 1899. Schletter'sche Buchhandlung.

[2] "Ueber die Gruppirung von Geisteskrankheiten," Danzig, 1863.

Wernicke has an easy task to show that etiology alone will always remain insufficient for classification. Acute hallucinosis occurs usually in alcoholism, but also without alcohol. The polyneuritic psychosis is essentially like certain senile disorders. Alcoholism may lead to: (1) the so-called transitory frenzy or pathological intoxication; (2) delirium tremens; (3) polyneuritic psychosis; (4) acute hallucinosis; (5) psychosis with anxiety, so frequent in alcoholics. Unless this is recognized and the forms are kept clearly distinct, we return to a general confusion and haziness. On the other hand, Wernicke recognizes the importance of etiology for the course of the disease, as is plainly shown in the parasyphilitic disorders classed as general paralysis.

Besides etiology, we must certainly consider the *localization and the nature of the disease-process*. Even in as homogeneous an organ as the liver, it matters much whether a focus of disease is involving the peritoneum or the environment of the vena portæ or the bile-duct; and this certainly holds much more strongly for a complex, highly differentiated organ such as that of association.

Wernicke applies the theory of specific energies—according to which any stimulation of a nerve is responded to by that nerve's specific sensation—to the entire system of associations. Every content of consciousness or concept depends on an altogether definite and limited set of nerve-elements; hence his psychiatry turns primarily to the disorders of the contents of consciousness as the first factor to be considered in all classification, because Wernicke places concepts in three groups: concepts of the external world, of the body, and of the so-called personality or individuality (the sum of individual memories of a person). Clinical experience shows, indeed, that the *chronic* disorders in which, according to Wernicke, the form and course of mental activity are not disturbed as such, are apt to affect principally one of the three fields, either chiefly the concepts of the outside world (including other persons), or those of the patient's body, or those of the patient's personality, and he therefore divides these mental disorders into allopsychoses, somatopsychoses, and autopsychoses, and the same principle is applied to the acute mental disorders in which, besides the derangement in the contents, there is a decided though variable affection of the form and trend of mental activity, the stream of thought.[1]

Concerning the nature of the *disease-process*, Wernicke is unitarian. Since, apart from general paralysis, mental disorders are either altogether functional (without an anatomical lesion) or simply depending on a primary degenerative process of nerve-fibers and nerve-cells, we have every reason to assume that there is really only one anatomical process, and all classification will therefore depend on etiology and localization alone.

How does Wernicke analyze his cases?

All our facts of mental disorders are primarily derived from the observation of motor reactions, since we cannot see into the mind directly. We call them all disorders of identification (deviations from doing or thinking or feeling the right or adequate thing in presence of a special fact to be identified), and we may distinguish disorders chiefly in the sensory sphere (sensory identification), or disorders chiefly in the intrapsychic sphere (intrapsychic identification, the thinking), or, finally, disorders mostly involving the motor mechanisms, disorders of "psychomotor identification." Within each of these three fields we may expect to class all derangements, either as afunction or parafunction, or hyperfunction (sensory, intrapsychic, or psychomotor). Yet there are a certain number of cases of acute insanity in which the disorder of the psychomotor reactions is so great that an analysis of the psychic content is really not possible, and we must be satisfied with the distinction into akinetic, hyperkinetic, or parakinetic ***motility-psychoses***, until, after the disappearance of the deepest perturbation, a distinction into autopsychic, allopsychic, and somatopsychic origin becomes possible.

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[1] Both Wernicke's idea of concepts and the division of conscious phenomena into content and form appear to me more logical than biological. The division of concepts, according to Wernicke, should point to certain localizations which are, however, quite hypothetical and by no means urgently demanded by the clinic, which keeps away from the stipulation of concept-centers. Wernicke's localization (outside-world, body, and personality) refers to topics of thought and by no means to localization in the brain. The division of mental activity into form and content, too, has also its drawback. We can, even in chronic psychoses, see that disorders of form might be made accountable for disorders of contents. The distinction of types of form, *i. e.*, of types of the stream of thought, is extensively used by Kraepelin, practically with disregard for the contents.

The fundamental assumption of such a classification of symptoms is that all mental disorders lead to "disorientation," a disorder of "secondary identification" *i. e.*, of the function of feeling, thinking, or doing something or other in an adequate way. Wernicke used the word in its broadest way, and was misunderstood by a reviewer in the "American Journal of Insanity," April, 1901 (who also mistakes Wernicke's acute hallucinosis for a delirium tremens, but otherwise does him full justice). Wernicke implies that all mental disorder shows in some *inadequacy*, or loss of proper bearings, and this "disorientation" leads to a characteristic affect, perplexity (easily confused with melancholia, as often by those who speak of "initial melancholia"). He therefore tries to establish in acute psychoses the extent of autopsychic, somatopsychic, allopsychic, or diffusely motor disorientation or lack of identification, and the extent of the accompanying affect of perplexity.

In explanation we may give the following hints. Wernicke distinguishes primary and secondary identification. The identification of the sound of a word as a sound is the primary, the grasp on the meaning is the mental appreciation or secondary identification; primary identification is simple sensation; secondary identification comprehends all the functions implied in mental grasp and elaboration. On the basis of his scheme of aphasia, he makes an anatomical hypothesis for the events in speech, in the form of a chain of nervous mechanisms, starting from the sensory projection field S (first temporal gyrus) and ending in the motor projection field m (Broca's convolution). The recognition of a word-sound as something known is a simple assimilation or primary identification. For the understanding, the mechanism of the concept must be roused, and this spreading is represented by s C, the "psychosensory path." For the purpose of adjustment of equilibrium in its relation to the rest of the person the concept C co-operates with a mechanism of reaction represented by the concept of aim or purpose P; and this, again, co-operates with a mechanism of enunciation m in the Broca convolution. The mechanism involved to lead from a simple concept to the concept of purpose C P is the intrapsychic complex, and the connection of the final concept P and the mechanism of enunciation is the psychomotor part of the secondary identification or association. Each of these components can show a minus or plus or perversion of activity, so that Wernicke distinguishes:

PSYCHOSENSORY SPHERE:	INTRAPSYCHIC SPHERE:	PSYCHOMOTOR SPHERE:
Anesthesia,	Afunction,	Akinesis,
Hyperesthesia,	Hyperfunction,	Hyperkinesis,
Paresthesia.	Parafunction.	Parakinesis.

This correlation of all psychic function with the function of speech is similar to what Stark arrived at (1838), who recognizes anoësis, hypernoësis and paranoësis, and abulia, hyperbulia, and parabulia, but it has the advantage of getting along without the hazy concepts of feeling, intellect, and will, by remaining on a broad biological though also hypothetical ground.

Looking back over this rather complicated nomenclature, one can hardly suppress the query whether the facts of observation might not lead to a classification of the data for more natural and less schematic reasons and with results which would be more easily manipulated and which would present themselves more directly.

Wernicke illustrates his method of analysis of cases. He first seeks to determine the sphere of concepts in which the patient is disoriented (or we might say inadequate), or at least threatened with disorientation. Then follows an analysis of the symptoms in accordance with the scheme of disorders of identification. Finally, etiology and course are considered. Thus delirium tremens is a psychosis with disorientation in reference to the outside world, an allopsychosis composed of: hallucinations of the various sensory domains, especially the optic and tactile (psychosensory hyperesthesia); illusions (psychosensory paresthesia); disorientation as to the locality (psychosensory anesthesia); unrest and delirium of occupation (psychosensory hyperkinesis); at times anxiety (psychosensory paresthesia). Taking it all in all, it is a complex of disorders of psychosensory identification, without independent disorder of intrapsychic or motor identification (the patient thinks and acts correctly in response to his sensory state). There is, moreover, in addition to the purely psychic symptoms and belonging in the field of organic disorders, an affection of the sensorium, which consists in dulness (*Benommenheit*) to an extent that varies at different moments and is influenced by external interests (the patient responds promptly when started out of his dream state). Furthermore, there are symptoms from the projection systems: Tremor, speech disorder, deviation of the tongue, paresis of the facial, etc. In mania and affective melancholia (as Wernicke describes them), orientation suffers chiefly as to the meaning and relation of the patient's own personality (exaltation or ideas of smallness, sin, etc.), and all the symptoms are referable to intrapsychic hyperfunction in mania, and intrapsychic afunction in affective melancholia. (Wernicke includes in mania simply the complex usually called hypomania; and under the term affective melancholia, cases of simple depression with feelings of inefficiency, retardation, etc.) This plan allows Wernicke to give a definite symptomatic analysis in about 50 per cent. of the acute psychoses, whereas the other half permits at least a provisional analysis. Even to the most experienced alienist new combinations are bound to occur; but in every case the system gives opportunity for provisional analysis and for combination of typical traits, which makes it easy for any observer to recognize identical cases or distinguish diverse ones.

Wernicke enumerates the following established types: (1) Delirium tremens, polyneuritic psychoses, and presbyophrenia are allopsychoses with allopsychic disorientation. The acute hallucinosis primarily shows merely allopsychic perplexity, and true allopsychic disorientation only in the characteristic paranoic phase. It is the type of an acute hallucinatory allopsychosis, as the chronic hallucinosis is the type of a chronic one. (2) Affective melancholia and pure mania and the corresponding circular psychoses are instances of autopsychoses; also paralytic megalomania and primary paranoia; and the cases with dominant ideas (as, for instance, the querulants) form a group of circumscribed autopsychoses. (3) Among the somatopsychoses we find the cases of purely intestinal or more generalized acute or chronic pure hypochondriacal psychoses (pure inasmuch as the allopsychic orientation is preserved, whereas hypochondriacal persecutory paranoia, an independent form of a chronic ascending psychosis, spreads into the allopsychic field and is to be classified as allosomatopsychosis). (4) The especially frequent cases of anxiety-psychoses, whose dominant symptom refers them to the somatopsychoses and makes them most closely related to the hypochondriacal psychoses, show nevertheless autopsychic and allopsychic perplexity, and, on the autopsychic field, also disorientation, so that they may also be classed with the combined psychoses. (5) Finally, Wernicke mentions the so-called periodic mania as a hyperkinetic motility psychosis (of usually menstrual origin), and the more or less pure cases of akinetic and parakinetic motility-psychoses.

In a number of other frequently occurring conditions, as, for instance, in what Wernicke calls depressive melancholia, our knowledge is not sufficiently definite to recognize them as disease-types; but the system makes it easy to classify them (with the autopsychoses). Concerning such uncertain types we still stand on the old ground of "*Habitualformen*," *i. e.*, symptom-complexes.

The idea that the character of symptoms changes frequently in the same patient is an old tradition. According to Wernicke, this is not true of the types mentioned above, and when it does occur (as sometimes on a hebephrenic basis, and especially in general paralysis), the case is best classed as a compound psychosis. There is certainly far less regularity in the sequence than was formerly held.[1] The doctrine of an initial melancholia has its origin in a confusion of melancholia with the painful affect of perplexity which accompanies almost all the acute psychoses at their onset and even later.

This free translation of Wernicke's article gives us a good introduction to his book, "Grundriss der Psychiatrie."

The first part of this consists of a psycho-physiological introduction and elucidates in great detail the above scheme of psychological analysis. Mental diseases are diffuse disorders of the organ of association, and can be dealt with on the basis of the scheme of aphasia. The adaptation of this scheme to psychology generally requires the recognition of a distinction between primary and secondary identification (see above), the dissolution of the "center of ideation" into concepts of various localization, and it operates with originating concepts (C) and final concepts (P—final purpose), as the beginning and end of the intrapsychic chain of activity, which, of course, consists of many similar links (see above classification of the scheme). All perception and all action are finally presented on this same scheme. S is merely the speech part of the receptive cortical field, and m the speech-portion of the emissive cortical area. The trans-cortical connections between the sensory and the motor cortex make all secondary identification possible. Primary identification (simple sensation) without secondary identification (perception and elaboration) would remain lost and not understood in the optic field as well as in the sensory speech-field. The third lecture deals with the memories, the fourth with their association (picture of the outside world, number of concepts, needs of causative explanations), the fifth with the body and its specific sensations, the sixth with reflex and spontaneous motion, and its dependence on sensations, the seventh with the consciousness of personality, and the eighth with the distinction of content and activity of consciousness, degrees of consciousness and attention and will, "*Merkfähigkeit*" (grasp), affects, and the equilibrium of ideas.

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[1] As in Kahlbaum's vesania typica and his definition of catatonia, in which Neumann's cycle of melancholia, mania, confusion dementia, plays such an unwarranted rôle.

I do not feel inclined to enter into detail upon Wernicke's general deductions, of which I gave an abstract in the "Psychological Review" (1895). The effort at a morphological presentation of mental functions (memory, association, etc.) suffers from the same drawbacks as all correlation of results of the working of a machine would, if we simply tried to correlate parts of the result with parts of the machine. Surely the pendulum of a clock plays an important part, but we can not identify it as such with the sound; but only its working with the occurrence of the sound.

The eighth lecture shows plainly that the morphological premises as such, after all, are unessential, since we do not and can not use them actually in the observations of the patients. As soon as Wernicke speaks of his observations of patients, the more practical issues take the lead, and there remain but a few assumptions which Wernicke attaches to his psycho-physical considerations, without, however convincing one of such a deep justification as he gives the morphological side of his hypotheses.

When we attempt to review the facts for psychic correlation or localization, we do not get far beyond what Wernicke calls primary identification. That permanent hemianopsia and cortical blindness are possible, and referable to destruction of the optic radiation and its cortical field of projection, is demonstrated. Concerning subdivisions of the visual field and any special relation of special localities to specific qualitative optic sensations (form and color and perception and memory), we know nothing conclusive. We know of cases in which vision seems possible with a visual acuity of up to ½ without a real appreciation of what is seen. In aphasia we find numbers and music reacting in a special way, and perhaps they must be referred to special localities; but why the recovery of words and diction follows practically the same laws as that of simple amnesia without organic basis is not explained on the basis of the ordinary view of memory-cells and the like. It is true that under the title of mind-blindness Charcot and Wilbrand have described peculiar permanent distinctions of modes of mental reactions. Charcot's case of loss of "visual images" (a merchant who had suddenly lost his visual memory and the ability to think in French) has certainly lost a rather circumscribed type of psychic reactions; but from this to a theory of localization of these functions, there is a long distance. One might well expect a subjective reaction where words are *forgotten* different from when they are supposed to be "destroyed," but even this difference is yet to be established, if it exist at all.

Wernicke's primary effort is to establish a tenable non-traditional symptomatology, and in this I see his greatness. His ideal would be attained beyond doubt were it possible to demonstrate that the symptoms depend upon known properties of the organ and their derangement by disease. This is, however, really fruitful of results only when we deal with accepted knowledge or accepted hypotheses. These of Wernicke are purely tentative, and probably more complicated than is advisable or necessary.

Wernicke bases his clinical discussions on a material of 5000 cases observed by him and his assistants during fifteen years. From this rich material he selects certain cases which appear most simple and lucid and open to thorough analysis. Only about 50 per cent. of the acute psychoses are covered; the other 50 per cent. appear as yet too complicated for a text-book, and there are continually cases added which must be classed as new types.

The feature that attracts me in Wernicke is the faithful adherence to actual observations of cases. There are no composite pictures of entire groups (in which it is so easy to juggle the symptoms a little for the sake of creating a clear picture), but always definite case-records on which he builds his discussions and conclusions. There is no ready-made coat to fit all cases covered by a traditional name, but a statement of the material and general features in a few cases and directions as to how to find and use these in others. There is no belittling individual traits in such descriptions, but there is an avowed plan, and much to help to distinguish the fundamentally important traits from the merely individual ones.

Wernicke starts—very wisely, it seems to me, though my reasons are derived from a purely practical standpoint—with the cases whose mental activity resembles in its course and form that of a healthy individual. In his language, these are cases with changes of the contents of consciousness notwithstanding normal or nearly normal course of mental activity. His first type is recovery without insight—"residual autopsychic falsification of memory"—a class which ought to be recognized by the public and by courts as not completely recovered; the second one is that of relative recovery, also without insight, but with additional delusions of explanation, of persecution, and perhaps consecutive ideas of exalted importance. In this group he shows cases of residual falsification of consciousness either in all the three fields of consciousness (a-total psychosis, auto-, allo-, and somatopsychic), or simply allopsychic, or somatopsychic—all merely cases of incomplete recovery, "old" or "chronic" cases. These residual derangements are to be distinguished from the mental disorders of chronic evolution, the chronic psychoses proper. *Psychosis* with him means a disease-process in activity; as soon as it comes to a standstill without recovery, the case enters the class of mere chronic *derangement*.

In this connection Wernicke demonstrates the importance of explanatory delusions (auto-, allo-, and somatopsychic) which arise on a basis of autochthonous ideas, or of motor symptoms, or even on a basis of perceptions of functions which, as such, are perfectly normal, such as the pulse or heart-beat. He also deals with hallucinations, ideas of reference, the retrospective explanations and falsifications of memory, and dominant ideas.[1]

Theoretically he postulates the occurrence of "*sejunction*," the local interruption of sets of associations, as an explanation of the lack of correction of delusions. The continuance of activity of the remaining functions, without realization and correction of the interruption, leads to all the secondary symptoms. Falsification of memory, and even hallucinations, are explained in this way. Owing to the local interruption or sejunction, no correction takes place.

For an analysis of this kind, paranoic conditions and the residual disorders following mental disease offer undoubtedly the best material. It does seem as if there were a block in the way of the formerly correct reasoning, and it is striking that in many patients the lapse is largely in the direction of one of the three fields distinguished by Wernicke, the auto-, allo-, or somatopsychic domains. But neither in actual correlation with happenings in the brain, nor in the question of localization, does Wernicke furnish evidence pointing to any actual gain in knowledge of fact concerning the brain as such. The autochthonous ideas are accepted as meteors, and the block-theory may satisfy many a student sufficiently to relieve him (perhaps to the disadvantage of the patient) of the duty of finding out how the strange thoughts came into existence, whether through some psychic trauma (as undoubtedly in many querulants) or through some other psychic process worth investigating with all the means at our command. The theory also winks at some favorite localizations of hallucinations. So long, however, as Wernicke remains within the bounds of psychical analysis on the basis of "personality," and speaks abstractly of dissolution or sejunction of personality through irritation or final interruption at certain points of his hypothetical path (sensory memory field—initial concept—final concept—motor memory field) we can only admire his genius and excellent metaphors. Imperative concepts he describes as an irritation with preserved continuity; autochthonous ideas as an irritation with partially dissolved continuity; hallucinations as an irritation with a blockade; and finally he tells us of simple sejunction without irritation—the mere residual uncorrected state. The interchangeability of autochthonous ideas and of hallucinations, and the possibility of persistence of hallucinations beyond the florid active period of the disease, is "explained" by sejunction.

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[1] These terms recur so often, are so useful, and likely to become lasting in our psychiatric phraseology, that a brief definition is excusable. *Autochthonous idea* signifies what the patient usually calls "a thought coming in some unaccountable way," strange, but not referred to an actual hallucination. An *idea of reference* is one where it is supposed that what others do or say refers to one's self, that trivial events or actions mean something in relation to one's self, etc. A *dominant idea* or unreasonable idea is an idea which has obtained to undue importance, usually through its emotional tone, and is exaggerated, as, for example, the ideas of the crank or querulant.

The acute psychoses yield to a great extent to the same principles of analysis. Wernicke sees the acuteness in the rapidity of evolution; acute psychoses are changes of the contents of consciousness which we see develop within a definite time. Such changes are usually marked by, and frequently obscured by, deep emotions and affects. The rapidity of the change stamps the attacks in another way. While in the chronic paranoic condition the lasting or fixed perversions of the *contents* of consciousness are uppermost, the morbid changes of *activity* of consciousness characterize the acute psychoses.

Wernicke offers no complete "general symptomatology." True to his principle, Wernicke presents his analysis in connection with the record of an actual recovered case, a very well-educated person who remembers and analyzes his symptoms excellently. We again meet with sejunction and delusions and hallucinations and autochthonous ideas. Their coherence and durability depend on a certain degree of connection, and on the possibility and extent of the mental funds. Wernicke shows us new sources of delusions: some that result from conclusions of analogy, some from inattention, and some that originate from morbid new-formation of associations. He discusses simple and compound hallucinations and the various theories of hallucination. For Wernicke a hallucination is due to the wiping out of the difference between a memory-picture and the picture of actual perception, which depends on perception-cells, the bearers of the organic feeling which belong to each actual sensation, and constitute the "somatic consciousness." Their activity gives the picture its reality, forces it into the center of attention (as is the case with all the derivatives of somatic consciousness). The point of attack of the irritation must be those localities which correspond to the memory-images and the effect spreads over entire "Merksysteme," or concatenations of (neuro-) psychic elements, and of the perception cells. Abnormal conditions of the latter *may* show in hallucinations. Thus, Pick observed a patient who formerly had had sensory aphasia, with auditory hallucinations of a distinctly paraphasic character; and another patient with a hemiopic defect of homonymous quadrants saw a defect of the corresponding sector in the hallucination of a face. The irritation in one hemisphere is probably sufficient; visions appear flat; voices seem much more real because the auditory center is one-sided, in the left temporal lobe. Certain forms of compulsory talk are probably a direct symptom of lesion of the left temporal lobe. But the irritation is, as a rule, of the nature of irradiation from processes of sejunction otherwise localized; for the same types of disease may occur with or without phonema (= auditory hallucination in words), and in one and the same case the contents may be expressed as a voice or come merely as a thought. The special localization of "memories" seems fairly well proved to Wernicke, and also the possibility of thought by means other than the left temporal lobe. The distinction between phonema and autochthonous idea (which usually comes in definite words, and therefore might be a symptom of irritation of the left temporal lobe) is referred to a hypothetically different localization of the sejunction.

The primary identification may also be affected in insanity (Wernicke includes in the mechanism of primary identification the activity of the perception-cells and the act of conduction to the memory-cells, the secondary identification is merely the transcortical conduction beyond the memory-cells): (1) In *hypermetamorphosis*, a term derived from Neumann,[1] meaning compulsory distractibility, due to increased irritability of the naturally dominating organic sensation, *i. e.*, actual vision, actual hearing, etc.; this sensitiveness may implicate more or less either hearing or vision; it always leads to distraction and disconnection of the stream of thoughts and it is a chief constituent of confusion (see, however, Wernicke, p. 221, 1). (2) In hyperesthesia of the sense-organs, chiefly in hysterical patients and at the onset or during convalescence. Only hypermetamorphosis is of importance, and it occurs, outside of agitated phases of general paralysis, chiefly in severe forms of confused mania and in hyperkinetic motility-psychoses. Another set of symptoms not strictly belonging in the group of disorders of secondary identification is of the same order as numerous automatic motor adjustments, like normal visual fixation, or reactions of defense in emotion, and in mental disease, jactations, motions of expression, attitudes of anxiety of variable localization, motor unrest or states of rigidity or akinesis, perhaps in analogy with what may come from pressure on internal organs. (Wernicke refers to the profound effects of pressure on the kidney, etc.)

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[1] In his excellent little "Leitfaden der Physiatrie" he speaks of the drift and stream of activity and thought as metamorphosis; too rapid a drift he calls hypermetamorphosis, whereas states of absorption and inactivity are called ametamorphosis.

Wernicke next deals with the concept of "disorientation." The disorders of identification bring about a condition which Wernicke calls disorientation, and according to the sphere of consciousness in which the provoking changes occur he speaks of autopsychic, allopsychic, somatopsychic, and motor disorientation. Wherever it sets in rapidly, it throws the patient into a vivid affect of perplexity and puzzle. That the hallucinations as such do not necessarily produce disorientation in the ordinary sense of the word, Wernicke shows in two patients, one of whom is an epileptic with most vivid hallucinations, but perfect recognition of people and place, etc. Among the various types, somatopsychic perplexity is most absorbing. It is possible that the localized feeling of anxiety is merely a type of somatopsychic perplexity. Also the *acts* of perplexity are described. The same case presented also peculiar motions which Wernicke calls pseudo-spontaneous motions (analogous to autochthonous ideas), unaccountable motions, felt as strange by the patient, also normally subject to explanatory delusions. The same patient attributes the initiative and reactive mutism merely to a certain difficulty of speech, not to any orders of the voices. He remembers, however, his surprise over certain circumscribed parakinetic and akinetic states, and that concerning them he had the idea that God put them on him. In a further lecture, Wernicke points to the dominant ideas and a specific type of special sensitiveness (compared to the sensitiveness of a sleeping mother to any noise from the child), called partial intrapsychic hypermetamorphosis. Finally, he gives his reasons for thinking that the form of both illusions and hallucinations is usually the expression of an affect, and that the form and content are determined by the dominant idea, or by the auto-, allo-, or somatopsychic or motor perplexity. After a thorough analysis of the rather complex case which Kraepelin would call a catatonia with remission, he begins the description of a series of types and goes on with their analysis.

Throughout, Wernicke aims at explanation of symptoms based upon the known functions of the organ. He makes good use of those excellent studies of his pupils on asymbolia, etc., and one feels that, at the same time, he is wide awake to the facts of the case, and in this respect at least perfectly true to his promise to put the observation of the case into the foreground and to adapt the method of interpretation to the needs. His descriptions are very faithful and the new nomenclature shown in them by no means mere form. There is no alienist who would not like to throw off many of the terms of descriptive cant seen in the case-records of hospitals; but the difficulty is how to replace them. Wernicke has certainly given us a splendid and useful help in his broadening out of the term disorientation (perhaps, better, inadequacy of attitude and reaction) to all loss of bearings in any sense whatever, as the fundamental trait of all insanity. And his next step, the recognition of "perplexity," is equally important as a topic of analysis where current nomenclature uses the terms "confusion" and "depression" without any further inclination to show what the trouble consisted in. His series of "dominant ideas," imperative ideas, autochthonous ideas, phonema, and "pseudo-spontaneous motion" is another clearing factor. The division of the realm of consciousness into facts referring to the patient's personality, to his body, and to the outside world and its living and non-living factors appears perhaps dogmatic, but is over and over again shown to be of fundamental importance owing to the totally different causal efficiency and symptomatic value of the three types—which, by the way, Wernicke does not mean to distinguish absolutely, at the expense of justice to facts, as he is quite willing to see transitions where they occur. And when we look at such a condition as delirium tremens, it is really striking how the limitation as a psychosensory allopsychosis opens one's eyes to a very distinctive feature.

The intense neurological interests of Wernicke, however, give his analysis a peculiar trait which has its disadvantages. The emphasis on localization, the preoccupation by "spatial" analysis, does not quite do justice to a field which is so eminently a stream of occurrences. However broad a place the localizing discussions occupy, in reference to the auto-, allo-, somatopsychic and motor disorders, they furnish little *nosological* result. The localization of the organic or corporeal component of sensations in the cells nearest the marrow, the consideration that hallucinations must involve memory fields and perception-cells, may supply serviceable suggestions for our phraseology as to brain-happenings; but as long as the all-important sejunction is somewhere else or anywhere, the localizing factor in the description is largely decorative, and, as I mentioned before, is apt to be deceptive and distracting. The sejunction is a formula. There are absolutely no facts for its analysis outside of what we observe in the development of the symptoms, and if we wish to go behind the symptomatic sejunction,—*i. e.*, behind the evidence of what we have in the way of facts in the form of a stream of events,—we merely find an improbable something in the form of an unknown lesion. We shall recur to this again. For the time, we repeat our expression of indebtedness to Wernicke, for having shown us a way to emancipate ourselves from traditional haziness, and for giving us some points of view which may have some value when our knowledge of correlation of brain and mind becomes a more useful and imperative problem than it is now, for want of facts.

The consequences of the "spatial" interests show clearly in Wernicke's presentation of the disease types. But he gives most excellent sketches of temporary states—just what one needs and wants to get at in actual life in examining a patient. His descriptions, as far as I am aware, have no equal in the entire literature of psychiatry. Everywhere we see a presentation of the palpable observations, and only that which is expressed by the patient and actually established by the physician is used for the analysis. There is no resorting to assumed anemias and hereditary predispositions, or to degeneracy, or even to better established etiology, as long as the examination of the symptomatology goes on; nothing is suggested as expected to exist "in such cases," as would happen if he started from set tradition; and since he sticks to his one case, he does not tire and confuse one with the statement that this and that symptom "may occur." In this respect Wernicke approaches the ideal exemplified in his colleague Foerster, to whom he has paid a most worthy tribute in admitting his great indebtedness to him. Wernicke goes so far as to say that, with his excellent method of examination and discrimination of clinical symptoms, Foerster would have established all the forms of amaurosis and their clinical value from a mere study of the *functional disorders*, even if the ophthalmoscope had never been discovered. And Wernicke drew the natural conclusion that in a field where an "encephaloscope" is not to be hoped for, an accurate study of symptomatology is all the more needed, furnishing, as it does, a means of getting out of what he calls "*die psychiatrische Misère.*"

The first type Wernicke describes is the anxiety psychosis, a type which is usually called agitated melancholia. This form of depression resolves itself chiefly into anxiety and perplexity. A perusal of his cases and their analysis and comparison with his "affective melancholia" and the conscientious description of a group of intermediate cases shows one at once his whole attitude, which is one *not bent on classifications*, but on understanding of the case. The anxiety psychoses are brought into close relation with the somatopsychoses and hypochondriacal disorders, forming a group of cases that usually appears in our statistics as "melancholia."

The next type is acute hallucinosis, followed by the sensory allopsychoses of which delirium tremens is a prototype; then comes the group of autopsychoses including certain hysterical states, certain forms of *Wahnsinn*, and imperative concepts. Next, Wernicke describes affective melancholia and simple mania; then hyperkinetic motility psychoses; then confused mania; further, akinetic motility psychoses; later certain compound forms; and, finally, as a group on an etiological basis, general paralysis. Finally he reviews other etiological groups, the course and outcome, and dissimulation and simulation, and the outlook in the direction of a pathological-anatomical understanding of mental disease.

We now turn to Kraepelin. The literary antecedents of his 1896 edition lie in the direction of the psychological experiment and of clinical psychiatry. His chief contributions to science are: (1) Studies of the influences of alcohol and other substances on mental activities, begun in Leipzig under the influence of Wundt. (2) Studies of a clinical-etiological character.[1] (3) Descriptive and analytical studies of disorders of memory.[2] (4) The various editions of his text-book (1880–1899).

The first edition of the text-book was the first extensive publication. It stands thoroughly on Wundt's grounds in its general part, and in the special part ranges itself at once with the works that furnish descriptions of *types* of events with little reference to individual cases. The relation to Wundt gave the book the stamp of the most up-to-date attempt at psychopathology, and it was recommended in my years of study as the best psychologically, while Krafft-Ebing was commended as most excellent clinically, although to my mind Schüle would probably have deserved the first recommendation for descriptive accuracy (it is, however, rather more true to nature and more complete than is good for the student). My personal reminiscence of the third edition of Kraepelin is that of a systematic and very readable work, but not of the kind that would have evoked in any one such a distinct feeling of contrast with other books and clinical teachings, as the last two editions have provoked.

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[1] "Arch. f. Psychiatrie," vols. XI and XII.

[2] "Arch. f. Psychiatrie," vols. XVII and XVIII.

The striking feature of the book was the marked change which occurred from edition to edition, and the gradual growth of what was at first a small compend to a work consisting of two large volumes and treating the subject from a totally different standpoint.

As the neurological study of the brain and the extension of neurological views into psychiatry mark Wernicke's work, so do psychological experiment with drugs and its method of investigation show, in a way, all through Kraepelin's work. The working of the minds of the two investigators is distinctly expressed by their chief interests in research. Their method in psychiatry is described by both as essentially clinical. We have shown it in our review of Wernicke, and Kraepelin claims for his whole recent tendency the distinctive term "clinical," with much justification (in a specific sense, which is best illustrated by the simile of longitudinal sections of the cases, *i. e.*, the study of the entire course of the disease is expected to reveal its true nature, rather than the mere cross-section, the temporary picture of just one phase or episode by itself). Wernicke starts from thoroughly analyzed observations of individual specific cases, Kraepelin from groups of cases. Only in his "Leitfaden," published in 1900, does Kraepelin give complete records of individual cases, and these are mostly analyzed from the point of view of course and outcome. There is nowhere a careful analysis of the "contents of consciousness," in Wernicke's sense of this phrase, but there is, on the other hand, a most conscientious analysis of the form of the *stream* of mental events, and of general symptomatology. He makes the *sequence* of events the main point, as in an experiment.

This emphasis of the stream of events points to a training obtained through the practice of experimentation. It supplements Wernicke's localizing analyses in a very important way, and (as the reading of Kraepelin would lead one to think) perhaps even replaces them essentially. Wernicke shows us the patient as representative of a group of symptoms in a topographic arrangement more or less in harmony with the problems of localization. Wernicke's conscientious adhesion to the facts does not fail to represent the symptoms as a chain of events; but the light is expected from grouping the symptoms topographically—either according to divisions of one's mental world, into auto-, allo-, and somatopsychoses, or according to localizing schemes in the brain (sejunction) or organism. It is a *chain* with the links simultaneously before us, not a *stream*, concerning which we would naturally be interested mostly in the direction and source and aim of the current. In Kraepelin the *chronological* side prevails, as it were; and from the fifth edition he says practically: It is the *sequence of events* that is of medical importance; the prognosis, the outcome, is the issue, and traits in the sequence of events are from the start the best material upon which to build nosological conclusions. With Wernicke, the existence of anxiety leads to the creation of an anxiety psychosis, in which he distinguishes allopsychic and somatopsychic, intestinal, ascending and diffuse forms, and a paralytic type—a group which includes acute, subacute, and chronic disorders of totally different nature, to be differentiated in each individual case. Anxiety appears as a fundamental symptom in almost every cross-section. Kraepelin divides these cases into totally different groups—into involution melancholia, into forms of dementia præcox, and formerly also paranoia, according to the nature of the course and especially the outcome of the disease.

In Ziehen, too, we find much reference to sequences, but in a much more restricted sense. While Wernicke divides the facts of observation into groups of practical importance, relatively free from traditional psychology, merely obeying the demands of his clinical sense and neurological habits, Ziehen groups them according to his psychology, which shines rather by its formal logical finish than by consideration of the special demands of medical experience. He places emotional life and intellect in two distinct categories; and mental disorders are classified into affective and intellectual forms, and in a more fundamental way into those without and those with intellectual defect (the dementias). This strictly analytical psychological plan has its clinical justification to some extent, and it employs certain principles of chronological sequence of development to some advantage. For instance, experience shows that it is quite a different thing whether depression sets in and is followed by delusions, or whether delusions set in and are followed by some depression. This is a principle of sequence quite extensively used. Kraepelin's observation of sequences refers, however, much more to elementary biological facts, and is, as such, independent of psychological systems. The most distinctive part is the description of *streams* of psychic events. That Kraepelin still adheres to Wundt's general plan of classification of psychology in his general symptomatology, I consider quite accidental, and of no positive consequence whatever, although I admit that it has certain distractive consequences. Without Wundt's psychology, Kraepelin would much more likely break through to a less formal presentation and shape his general part into more profitably arranged matter.

For years Kraepelin has been at work studying the effect of drugs and of fatigue and other influences on the trend and form of mental reactions. For this purpose he has systematized experimental tests, from which he derives his information in a number of directions which are really more modifications of general biological traits than topics of logical analysis of psychological products. His data refer mostly to the amount and efficiency of work, as shown by the ergograph experiment, additions, reading, association-tests, and the effects of practice and fatigue.

These data play a great part in the examinations of his cases. The stream of thought and its deviations from the normal are indeed as prominent with him as the classification of the contents of consciousness and the disorders of orientation are with Wernicke. The large group of manic-depressive insanity is made up of cases in which symptoms of inhibition of thought or of flight of ideas, stand out characteristically beside emotional and motor changes. This group is the best founded entity of the Heidelberg school. Clinical experience shows that the traits described mark conditions which do not pass into dementias and which stand out for other reasons (belonging to the typically "clinical" trend of thought) as a disease-entity clearly distinct from the large group of dementia præcox.

Kraepelin had publicly declined the method of Ziehen and expressed his disbelief that what we called above "logical psychology" could be of any real value in psychiatric nosology. He especially criticized Ziehen's paranoia group in his review in the "Zeitschrift für Nervenheilkunde," as bringing together essentially chronic conditions and acute psychoses of a totally different stamp, simply because the disorder happened to be in the field of the intellect, as Ziehen defines it, and because the cross-section has certain similarities.

The emancipation of *manic-depressive insanity* from the rest of the acute psychoses brought about an interesting revolution. It implied the creation of a group on symptomatic foundation and at the same time with a definite prognostic forecast. This coincidence, and the practical advantage derived from it, had so far been the distinction of only three forms of disease—of general paralysis, of paranoia (in the sense of a chronic delusional transformation), and of senile dementia. The result of singling out manic-depressive insanity was that climacteric melancholia had to be separated from manic-depressive melancholia, and that a certain number of "acute psychoses" were grouped with the usually subacute "psychoses of deterioration" on account of their usual outcome, although in a certain number of them deterioration was not sufficiently certain. The exhaustion psychoses remained, but in practice they were found less frequently because many cases formerly classified as such, showed the distinguishing features of manic-depressive insanity, or of psychoses of deterioration.

All through, general paralysis becomes the one paradigm of a mental disease, and the traditional forms are called symptom-complexes, unless they fulfil the requirement of definite etiology, course, and outcome. From the point of view of mental symptoms, as such, general paralysis gives indeed the greatest possible latitude. What distinguishes general paralysis had to lie, as most alienists say, either in the addition of the projection symptoms or neurological symptoms which characterize general paralysis, or in the differences of mental symptoms, inasmuch as on close observation there is, after all, in paralytic paranoia, paralytic circular insanity, or paralytic delirium, a certain something which usually makes it possible to distinguish them from simple paranoia, manic-depressive insanity, or delirium. Kraepelin seems to favor the latter view in his effort to show that even from the mental symptoms one can make a diagnosis of general paralysis, and he emphasizes just these distinguishing features. A further step was that other conditions in which it would be possible to draw conclusions from special marks of the symptom-complex (the cross-section), as to etiology, course, and outcome (the longitudinal section), could be also classed as diseases—and only these.

The relatively small material of the Heidelberg clinic, and the fact that it is fed by a district which is easily accessible and furnishes extensive records of the patients' families and relatively uniform conditions of life, render it in most respects ideal for an extensive study of what the patients were, are, and prove to be, in the subsequent years of their lives. These are points which Kraepelin made all-important as a test of his views. Few investigators are as favorably situated and, indeed, few were prepared to bring Kraepelin's views to a test on their own material. Moreover, the sense for the distinctions of the stream of thought which Kraepelin makes is not easy to acquire. The reception which he found was necessarily dependent on other factors as well; but nobody can fail to recognize with Wernicke that his presentation has found the greatest appreciation, especially among the younger generation of alienists. Whether this is due to their inexperience or to the tickling of the palate of beginners with promises of prognostic precision, or whether it is due to the dogmatism of older alienists and a sort of pessimistic vanity on their part, it will be our task to find out. To me, personally, it has seemed from the beginning to be the most stimulating guide available for hospital-physicians, an excellent ladder on which to climb out of the indifference of traditional statistics and the apparent hopelessness of even our best efforts at accomplishment. Nobody in this country has shown as clearly as August Hoch how valuable Kraepelin's principles are for one sensitive to deviations from the normal stream of thought. With the greatly varied and much larger and consequently less thoroughly studied material of the Worcester Insane Hospital, I found many difficulties, but Kraepelin's principles proved most valuable aids in my efforts to collect material upon which to base my own psychiatric views.

I have frequently been struck by the difficulty which many alienists have in their attempts to assimilate and correctly rate the new acquisitions. If we analyze briefly what Kraepelin says of the differential diagnosis of various disease-types, we see everywhere strong emphasis laid on certain fundamental traits, many of which have been casually mentioned by former writers, but which are here brought into a closer connection; instead of being mentioned as accompanying features, they become distinctive traits of entire groups. These groups are created for a definite purpose, which is largely prognostic, and Kraepelin shows that distinctive traits are available and deserve to be pushed to the front. What puzzles the reader is that the terms which they find are in part old acquaintances to which little meaning has been attached, and which are not very coherent from any point of view of localization, nor always clear in their origin. It takes close inspection to see that the old vague terms must be understood with new definitions and restrictions. Much of the difficulty comes from Kraepelin's aversion to assimilating the views of other men in a more than passing way (the remarks on Magnan, vol. II, p. 199, are an exception). It would be so much easier to understand him clearly if we knew how he deals with what we assume to know clearly ourselves or see as accepted property of the literature. Unless this is done, the uninitiated reader will approach the task and discover in the writings of Kraepelin chiefly those points to which they are positively or negatively sensitive. Kraepelin certainly does not help one to start from generally accepted views. There is, moreover, a decided dogmatism, perhaps more in certain followers of Kraepelin, than in Kraepelin himself, a non-compromising attitude which makes it difficult to take in his very sweeping changes of the tenets of psychiatry.

Perhaps too much is expected from Kraepelin's method. Wernicke frankly refuses to attempt a classification of cases, but offers a description and an analysis of manifestations such as everybody will be able to recognize again and to verify. About 50 per cent. of the acute cases yield readily to this plan. Kraepelin, however, has larger groups which would seem to offer a greater guarantee that most cases can be classified. He does not insist enough, as a cautious teacher should do, on the existence of an irreducible residuum, such as all honest statistics should make provision for, and his followers are apt to become imbued with the idea that while it may be difficult to classify many cases in the current official statistics, Kraepelin's standpoint allows of classification in practially every case. I plead guilty myself to such a tendency in the past; but I make the requirements of official statistics responsible for this, not Kraepelin's teachings. In order to do him justice, one must remember remarks like the following (vol. II, p. 2): "While it is, therefore, necessary to leave out of account—for all time to come—a smooth classification of mental disorders of the nature of Linné's botanical system, and even to feel resigned, at least for the present, concerning the establishment of scientifically well-founded types, there is nevertheless a practical need for at least an approximate grouping of the empirical raw material. Its value will be all the more lasting the less it is influenced by preconceived opinions in a sober digestion of facts." Or vol. II, p. 205, in the final discussion on dementia præcox: "Whether dementia præcox, in the scope given it here, presents one disease, must remain doubtful for the present at least. It would comprehend about 14 or 15 per cent. of all the admissions," etc.

We also find repeatedly the admission of great difficulty in differential diagnosis and considerably more caution than could be inferred from some of his followers. The *danger for the purity of his views* comes from the admittedly practical aim and from the fact that prognosis is made such an important part of the diagnosis. The didactic maxim is no longer chiefly that of sober analysis of what comes up in the empirical field, and a division into what is understood scientifically, and what not. The question to be solved is one of prognosis, which must anyhow be answered at least tentatively in every case. To be sure, this most stimulative feature of Kraepelin's teachings can be used most profitably. It is well to make good use of our natural inclination to formulate a prognosis and to say why we surmise the one outcome or the other, and that, instead of vague generalities, we should aim to record as far as possible traits which can be demonstrated in the patient at any time, or at least facts accurately written down in the history, not general phrases and intangible intuitions as the foundation of prognosis. This is part of the eminently didactic and progressive turn he gives to all work done according to his maxims. But every one knows how many cases there are in which a prognostic forecast is impossible, or at least mere guess-work, and in these a conflict begins between compulsory classification and classification of what may well be considered as ready and fit. I gladly acknowledge my deep indebtedness to Hoch, and later to Wernicke, for my gradual re-emancipation from a one-sidedness for which I am sure Kraepelin could not have been altogether accountable. By trying to fit every case, one comes to a method of diagnosis by exclusion which is not worthy of the term of good psychiatric work. It must be said, though, that Kraepelin does not warn us sufficiently against the expedient into which one is forced by an exaggeration of the importance of the prognostic element of the disease-concepts. (The prognostic element has been again extolled by Aschaffenburg quite lately.)

The purest expression of a truly psychiatric use and elaboration of clinical principles, in the sense of Kraepelin at his best, is undoubtedly Hoch's, both in his general article on the methods of psychiatry, and in his monographic articles on mania and on melancholia. He sets a splendid example for the requirement of positive traits for differential diagnosis and for the necessity of setting them forth in unmistakable observations of actual cases, and not in vague generalities. As soon as this is done, a group of doubtful cases is created. The central group of known cases is kept clear of admixtures which disturb the outline unnecessarily, and the central group stands for what it is worth. In Kraepelin's text-book there are only descriptions of groups. They are very striking, but they do not reach the ideal of composite photographs. The descriptions contain many individual details which would be submerged in a composite photograph. So much is put forth in one chapter that recurs in a similar way in another, that Wernicke's criticism of haziness of distinction becomes quite justified.

One can not fail to see in Kraepelin's work the slight stress placed on localization of the contents of consciousness, excepting as far as they are demonstrative of certain general traits. The condition of the sensorium (*Besonnenheit*), the coherence of the stream of thought and the forms of defect, the extent of a plastic grasp on the situation, and its actual utilization, the condition of acquisitive and reproductive memory, the existence of odd and strained attitudes as compared to free or inhibited or otherwise defective determination of activity—these are rather broad and biologically important traits which always recur in the differential diagnosis. A distinction into auto, somato-, and allopsychic disorientation or delusions, etc., is not attempted or found necessary. Analysis of types of delusions and hallucinations receives attention only in the theoretical part. The symptom-complex as such is no longer interesting through its analysis as such, but through its position in the entire evolution of the disease. And under this influence one's patience for the analysis of symptom-complexes or case-records depends altogether on whether the evolution and the outcome are properly reported. This, to my mind, is a step in advance, bringing psychiatry forward into the line of natural interests of the physician. We come down to plain questions.

We approach now a very important point. Kraepelin subordinates the symptom-complex to a broader clinical, or we might more properly say to a more simply medical principle—that of evolution and outcome. To what extent has he created "diseases," or, at least, *to what extent are his diseases distinct entities from the point of view of pathology for pathology's sake*, not pathology with undue reference to practical aims?

The fifth edition has much greater pretensions in this respect than the sixth. In 1896 Kraepelin practically recognized merely:

I. The group of exhaustive and infective origin (post-febrile and collapse deliria; amentia).
II. The group of intoxication (alcohol, morphin, cocain).
III. The processes of disorder of metabolism (myxedema, dementia præcox, general paralysis).
IV. Insanity due to cerebral disease.
V. The disorders characteristic of the period of involution (melancholia, senile dementia).
VI. Constitutional disorders (manic-depressive insanity, paranoia, disorders accompanying the neuroses, idiocy).

A number of these categorical steps which I criticized in part in my review in the "American Journal of Insanity" (Oct., 1896), as undemonstrated and too peremptory claims, have been changed in the sixth edition. The idea of diseases of metabolism is less prominent and the term constitutional psychoses is omitted; moreover, the author found many minor changes necessary. In its present shape the most dogmatic claims are dropped and the whole is more in keeping with the quotations from Kraepelin in an earlier part of this chapter. The question whether these headings are meant to describe disease-entities is no longer as definite an issue as when Kraepelin first boldly made the claim; and we are altogether in harmony with him if we ask simply: To what extent do the groups *approach* the requisites of a definition of a disease-entity?

Here we meet first the burning question: *Is it justifiable to consider general paralysis the paradigm of mental disease?*

In this question Wernicke and Kraepelin take extremely opposing views. Wernicke calls general paralysis an etiological disease-group. True to the facts, he admits a parallelism between brain-lesion and the symptoms of the lesions of the projection-system and of defects of the nature of dementia, whereas the great variety of the mental pictures is put down as yet unexplained and uncorrelated except by conclusions of analogy. The mental disorders are: (1) the expansive autopsychosis (the megalomania of older writers); (2) paralytic mania (not always distinguishable from pure mania, and frequently passing into the expansive autopsychosis, or at times into a remission); (3) affective melancholia (depression with feeling of insufficiency), rarely pure, usually with an admixture of delusions of reference or with symptoms of an anxious psychosis; (4) depressive melancholia (akinetic, or hypokinetic depression), also often with admixtures of delusions of reference; (5) among paralytic allopsychoses we meet frequently a paralytic delirium, which may occur at any phase of the disease, and at times resembles closely a delirium tremens, but more frequently a stupor with disturbed sensorium. Or the presbyophrenic complex occurs, or a chronic hallucinosis, or dream-states, resembling epilepsy, although but rarely, whereas the anxious psychosis is not infrequently of paralytic origin; also hyperkinetic and akinetic motility psychoses (akin to catatonia) are represented.

On this ground one may say that general paralysis may show itself as dementia after a simple psychosis; but usually the latter shows "paralytic" admixtures from the start or passes into other forms (as a mixed psychosis).

Nobody will deny the correctness of these analytical considerations. It does full justice to the absolute necessity of leaving the diagnosis in suspense in a number of cases with indefinite physical and mental symptoms, and to the cases which seem to favor the old view that general paralysis may be a complication of any mental disorder, an occasional surprise in the course of a paranoia or mania or stupor, etc., examples of which every alienist has certainly observed, and which should be remembered in the estimation of accuracy of a diagnosis of a simple psychosis. It also encourages a hope of Wernicke's that perhaps it might become possible to use the lesions present in general paralysis, for a determination of what parts are affected in definite types of psychoses. Without a very strenuous effort to differentiate these types of psychoses, which may be symptoms of irritation as well as of defect, we can not expect to use the material for a general anatomical explanation of mental disorders.

Kraepelin compares this with a custom of the past which gave emphasis to the mental symptoms and qualified the accompanying nervous disorders as simple "complications." To him, general paralysis is a progressive dementia with heterogeneous nervous phenomena of irritation and paralysis, leading to a complete annihilation of the mental and physical personality. "This process is accompanied on the mental side by more or less prominent symptoms of irritation, of excitement, depression, and by the formation of delusions of greatly varying content. The *common stamp* in all these derangements is a characteristic mental weakness," etc.

Kraepelin then proceeds to analyze the fundamental traits—gradual deterioration, disorders of sensorium, exhaustibility, disorders of acquisitive and reproductive memory, especially for dates, poverty of the stream of thought, filling up of gaps by confabulation, lack of judgment concerning the illness, etc., increased irritability, and later dullness, change of character and behavior; and, finally, he describes the physical symptoms accompanying the disorders of mentality. He gives up an attempt to do justice to the multiplicity of mental symptom-complexes, and merely sketches four groups—the depressed, the expansive, the agitated, and the demented.

From practical and especially didactic points of view such a presentation commends itself as eminently correct and useful and much less circumstantial than Wernicke's. One may even go further, and say that it demonstrates lack of clinical balance in those who put so much stress on formal traits of insanity as to obscure the chief points at issue for the beginner and the practitioner. One may well say that for research, Wernicke's accurate standpoint is highly commendable, and even essential, but that, for the needs of practice, the accentuation of the decisive features is what is wanted. This is undoubtedly the most important lesson taught by the consideration of general paralysis and fully put forth by Kraepelin. The next consequence is the question: Does not the same hold for all other mental diseases? Is there not too much energy spent on the analysis of symptom-complexes from fruitless points of view at the expense of that which interests practitioners? Or has the time not come yet for a radical change?

The bold stroke of 1896 has reappeared in 1899 in a decidedly mitigated form, and we may well ask: Will a future edition continue in this relative retreat and strike a level more in harmony with the average consensus in psychiatry?

Wherein lies the strength of Kraepelin's position?

The most suggestive and refreshing impetus of Kraepelin's departure does not lie quite as much in the creation of pathologically analyzed and unmistakably understood types, but in the spirit of emphasis of that which is medically most important. That impetus will never be lost by one who has ever been affected by it. Descriptions of symptom-complexes without descriptions of evolution and outcome will always be unsatisfying, and we crave for complete accounts of individual cases. But the dogmatism of disease-entities and of definite disease-processes underlying all their symptoms seems to meet a certain antagonism.

Let us begin with *manic-depressive insanity*, and give Kraepelin the field:

"Manic-depressive insanity comprehends, on one hand, the entire domain of so-called periodic and circular insanity, and, on the other, simple mania usually distinguished from the above. In the course of years I have become more and more convinced that all the pictures mentioned are merely forms of one single disease-process. It may well be that in the course of time a series of subtypes will be made, or even that certain small groups will be again separated entirely, but if this happens it will, as far as I can see, certainly not be determined by those symptoms which are now commonly put into the foreground. The reason for my taking this position is the experience that in all the disease-types mentioned certain fundamental traits recur in the same shape notwithstanding manifold superficial differences. Whoever knows the fundamental traits will, with due consideration to certain practical difficulties, always be able to conclude from a single phase that it belongs to the large circle of forms included under manic-depressive insanity. In this way a number of points are gained for the special clinical and prognostic meaning of the case. On the other hand, it is, as far as I can see, quite impossible to find any definite boundaries between the single disease-pictures which have been kept apart so far. From 'simple' mania, the numerous cases with two, three, four attacks in a lifetime lead over quite gradually to periodic forms, and from these we reach circular insanity, through those cases in which a more and more marked initial or terminal stage of depression gradually complicates the pure picture of mania, or in which the long series of maniacal attacks is unexpectedly interrupted by a state of depression.

"There is no alienist, and, I am convinced, there can not be one, who would be able to recognize from the temporary picture alone whether any case is a simple mania, or a periodic one, or circular. However different the picture of the excitement or the depression may be as such, it tells us about as little for the special clinical interpretation of a case as, for instance, do the various types of paralytic excitement or depression for conclusions as to the probable further course of dementia paralytica. There are, of course, in either case, certain general statistical rules, which, however, are very deceptive for the individual case, and therefore can never be of use for the delimitation of real diseases.

"Moreover, we not infrequently see in one and the same case the most different temporary pictures follow one another. All the manifold types of manic excitement may replace one another in one and the same and in successive attacks, in simple as well as in periodic mania, and in circular insanity. They appear therefore as *equivalents*, as an expression of quite closely related fundamental conditions which pass into one another quite naturally. In the superficially manifold and temporary pictures of epileptic insanity, dementia præcox, and general paralysis, we always turn to the definite fundamental disease-process. There, all recognized pictures can develop one after the other in one and the same patient. In a similar way there is in manic-depressive insanity a definite set of clinical types which substitute one another, without our being justified in tracing the innumerable varieties of form of the disease-picture to essentially different fundamental processes. On the other hand, we are quite in a position to class even the smallest fragment of the course of an attack which belongs here as belonging to the large frame of manic-depressive insanity. There are no bridges leading over to the other groups of mental disorders, with the exception, perhaps, of degenerative insanity.[1] For all these reasons I am led to conceive the clinical types of manic-depressive insanity as a unit, and to represent the individual temporary pictures and types of evolution as individual expressions of a common disease-process.

"*Manic-depressive insanity*, as its name indicates, takes its course in single attacks, which either present the signs of so-called manic excitement (flight of ideas, exaltation, and overactivity), or those of a peculiar psychic depression with psychomotor inhibition, or a mixture of the two states."

What actual help does Kraepelin furnish us to distinguish his manic-depressive cases from the other acute psychoses?

First: What are the common signs of manic-depressive equivalents?

Second: Which are the most important equivalents?

Kraepelin considers attention and comprehension, the sensorium and orientation, hallucinations and delusions, the stream of thought, the mood and the general effect on the motor life, its trend, efficiency, exhaustibility, with special reference to speech and writing—mostly points which are open to comparison with experimental material and in many respects open to test-experiment with the patient. It is hardly necessary to give more than a meager summary, since an abstract is available in English.

Attention and comprehension suffer in all but the slightest cases. In excited phases it is chiefly an extraordinary distractibility and insufficient grasp and elaboration of the rush of impressions, showing in overexcitability in some directions and insensitiveness in others (exposure). In depression, distractibility may persist, but the trouble with the elaboration and comprehension of impressions is much more prominent.

The *sensorium* is always slightly affected in severer cases. This shows in slight disturbance of orientation, especially concerning persons; in depressions and stupor the disturbance is especially apt to be pronounced, even to the depth of a lasting dream-state.

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[1] Which, according to vol. II, p. 529, includes the general neuroses, manic-depressive insanity, certain forms of imbecility, and perhaps also paranoia; like all the products of the theory of "degeneration," an etiologically rather vague conglomeration in which constitutional peculiarities play the important rôle, whether truly "degenerative" or not.

Hallucinations are not infrequent, usually of the nature of illusions, or actual, as especially in the depressive and stuporous phases. Delusions are very changeable, but sometimes they are held and elaborated (ideas of grandeur, persecution, jealousy, fear of poisoning). In depressions there occur especially ideas of sinfulness, of persecution, and of hypochondriacal nature. Their frequently fantastic nature, reminding one of general paralysis or senility (especially the hypochondriacal ideas), is held up as evidence of the view that neither the content of delusions nor their mere occurrence allows of any conclusions concerning the nature of the disease-process. A certain insight into the disease is usually present, but excitement or depression is usually referred to personal or external causes.

The *stream of thought* shows the most characteristic disorders. Mere habit and the greater ease in yielding to momentary impulses get the better of what would be demanded by actual circumspection and aim; and a flight of ideas is the result. There is no far-sighted leading purpose; the resulting incoherence is, however, not lawless. The unrestrained yielding to all sorts of impressions and whims even creates the appearance of increased quickness of perception. In depressions the patients may actually complain of all kinds of thoughts passing through their heads and of the resulting confusion; there is, moreover, difficulty in disposing of the thoughts, and usually an actual inhibition of thought, so that the patient may appear very limited and even demented. Even in the excited phases the "acceleration" of ideation really only covers up the slowness of association owing to the flightiness and instability of the links of the trend of thought.

The *mood* is a decided feature; exaltation or irritability, or true depression, and not infrequently a mixture of these traits.

The most striking disorders belong to the *psychomotor sphere*. The patient is all activity, yields irresistibly to every impulse; but in distinction from other forms of motor restlessness, manic restlessness can almost always be traced to some moods or purposes, playfulness, etc. The condition is easily exaggerated, and the ready incitability is perhaps the fundamental trait, together with insensitiveness to fatigue. The part played by speech, the tendency to drift in the direction of alliterations and rhymes, is especially important owing to its influence on the flight of topics in which connection and leading thought are lost. In the writing, too, the trivial signs of activity get the better of legibility and sense.

In depressions the very opposite occurs in the form of psychomotor inhibition. Indecision, difficulty or inability to make up one's mind, show especially in expressions, speech; and the mere hesitation at the start leads over to hesitation and slowness and weakness in the execution. External stimulation or danger may overcome the inhibition; but in deep depression or stupor even these may fail.

Inhibition and facilitation of voluntary impulses are, however, hardly as great contrasts as would seem at first sight. They are so apt to pass abruptly into one another, and so often they occur in a peculiar mixture, that the characteristic variability of the voluntary responses is, after all, one of the fundamental traits of this entire disease-group.

For didactic and practical purposes, Kraepelin classifies the material of pertinent observations into three main groups—the preeminently manic, the preeminently depressed, and the mixed states.

He gives excellent pictures of the slightest forms of excitement (*hypomania*), of severer forms (*mania*), and the forms with transitory or lasting confusion and greater prominence of hallucinatory and delusional productivity—all with characteristic moods and flight. Finally he gives a picture of the usually much shorter *delirious attacks*, of a little over three or four weeks' duration, with a very rapid development of a deep dream like clouding of consciousness, complete disorientation with numerous hallucinations and confused delusions of greatly varying color, so that the patient drifts rapidly from fear and anxiety to elation and ecstasy, or into dull indifference. Senseless motor excitement, beyond the possibility of fixation of attention, extreme flight of ideas and distractibility, vary with times of confused dullness; there is no sleep; rapid decay; frequently tremor of the whole body and lively reflexes. Remnants of delusions and anomalous mood may outlast the excitement and confusion, and diffidence, irritability, and perhaps also slight flight of ideas may persist for several weeks. Usually the events of the psychosis are forgotten completely. The duration of the manic forms is longer, usually months, not infrequently two or three years, especially when there are manifold delusions and only moderate transitory exacerbations of excitement. The termination is frequently a slight depression before complete recovery takes place. After severe attacks in some cases it seems that a certain lack of judgment, lack of insight into the disease, irritability, and moodiness may be a permanent residual.

The *depressive attacks* show similar degrees. A feeling of difficulty of concentration and thought, subjective insufficiency and dullness, a hopeless attitude, lack of interest and fear of the future, occasionally imperative trains of thought (a symptom common to types of degenerative insanity), indecision, and inactivity mark the lighter cases. Some cases may pass into a real stupor in which the patient grasps nothing, shows hardly any affect except perhaps slight perplexity, and a few attacks of anxious self-defense. Catalepsy and aboulia, or planless resistance, may be met with. The memory of the attack is very hazy.

In a second large group delusions, especially of sinfulness and persecution, are superadded to the disorder of thought and psychomotor inhibition, and also occasional hallucinations of sight, hearing, etc. Or hallucinations and delusions of persecution may set in, as in alcoholic hallucinosis, with perfectly clear sensorium, and complete orientation; there is, however, a certain monotony in the complaints and a rather rapid giving out in thinking, speaking, or writing; the mood is one of discouragement, or of dull staring, or of despair with monotonous wailing. Usually a certain psychomotor inhibition is demonstrable. In other cases a deep stuporous cloudiness of consciousness may appear; over the disconnected but greatly varied and often absurd deliria and hallucinations, the patient becomes inaccessible, mute, only showing the strange mental state by a tense anxious expression and resistance against influences, or by casual odd attitudes or motions, or by an occasional suicidal impulse; or at times they produce fragmentary utterances. Some of the illusions may persist during convalescence.

The third "*mixed*" *type* is theoretically and practically very important, and its description by the Heidelberg school a most creditable achievement. Kraepelin refers here to those unaccountable changes from exaltation to depression and vice versa, and to the actual mixture of simultaneous manic and depressive symptoms, which are either transitory (jokes or other signs of exhilaration, or of facilitation of voluntary reactions in the midst of a depression, or depressive and anxious moods in the manic) or persistent, as, for instance, the cases in whom the manic symptoms coexist with inhibition or depression with excitement throughout the greater part of an attack. He describes forms of exaltation with decided dearth of ideas, slow and inaccurate attention with times of very inadequate response. With it all the patient is cheerful, laughs, is excitable, makes fun; but the restless activity is limited to a few pranks or occasional outbreaks. In extreme cases the patient appears almost stuporous (hence the term "manic stupor"). Other cases are exalted, but mainly fault-finding and irritable. These conditions are not infrequent during the transition from manic to depressive states.

In other cases depression is accompanied by a veritable flight of ideas in the expression of delusions of sinfulness and despair; or the patients merely speak later of the rush of ideas. These episodes occur at times in the transition from depression to mania.

If we add to this, that the *physical condition* shows certain definite relations to the mental disorder, that cause and nature appear to be mainly constitutional, that the course is episodic and only in 4 or 5 per cent. consisting of continuous circles, and that only in few cases slight residuals develop (only the short attacks which recur very often seem to tend to a certain deterioration)—we have a fair summary of the group.

After his description of the forms, Kraepelin resumes the discussion. He points out the great variability and relative triviality of the *cause* of single attacks, and the often surprising independence of the whole group from external influence, and insists on the constitutional feature in their etiology.

Kraepelin's argument appears very convincing, unless there should be too many transition cases to the group of dementia præcox. There also may be a question as to whether the periodic mental disorders are really all manic-depressive. It certainly demolishes, effectively in the eyes of an impartial alienist, the old dogma concerning certain traditional distinctions, and pushes to the front matters of greater medical interest.

We must now take up another large group of Kraepelin's, that of *dementia præcox*. As will be remembered from the quotation heretofore cited, it is a provisional group, originally described as a disease of metabolism, together with myxedema and general paralysis. Referring to our previous remarks, we recapitulate Kraepelin's belief of 1896. He realizes (fifth edition, p. 414) that he goes beyond the facts established by research by assuming that in all these diseases the cortex is damaged by poisons produced by a morbid constitution of the fluid parts of the body owing to the derangement of certain organs which have a special influence on metabolism. "This conception proved, however, so fruitful in myxedema and cretinism that its further application is warranted on the mere fact that the clinical coincidence with these forms is very obvious in the chief characteristics."

This assumption has been dropped for lack of sufficient evidence,[1] and the term "processes of deterioration" is abandoned because deterioration is the key-note of general paralysis and of senile dementia as well, and they might be understood as included under that term. We shall also see that Kraepelin acquiesces in the generally accepted distrust of prognostic principles at least in melancholia (which is a disease of variable prognosis, and therefore a different sort of an entity from Kraepelin's other disease types, one not consistently created from the prognostic standpoint); and we may well be anxious to learn why deterioration should be considered the chief characteristic of a special group. Is it a deterioration such as would never result from a disease like melancholia, or any other "simple" disorder? When defining dementia præcox as a "series of disease-pictures, the common feature of which is the termination in a special kind of state of mental weakness," Kraepelin adds: "It seems, however, that this unfavorable termination need not take place without exception, but it is so uncommonly frequent that for the present we still prefer to maintain the term in use. Perhaps other terms, such as that of *demenza primitiva* of the Italians, or *dementia simplex* (preferred by Rieger), are even more appropriate. According to the clinical and anatomical facts known so far, I cannot doubt but that we deal here with severe damage to the cortex, as a rule but partially recoverable at best. Whether the disease-process is the same throughout must be left in uncertainty at the present time." Further: "The variability of temporary pictures (symptom-complexes) which we observe in the course of dementia præcox is very large, so that, on superficial observation, the inner connection can often be recognized only with great difficulty. We find, however, certain fundamental derangements over and over again in a more or less explicit form; most purely, it is true, in the terminal states in which the more or less accidental and transitory accessory symptoms of the disease-process have receded behind the lasting and characteristic changes of the mental life."

This elevation of the "outcome" to a nosological criterion is a very radical step. *A priori*, many objections on general medical principles revolt against it. But all objections will fail if the empirical result is in harmony with the claim. In an empirical science we are far less concerned about the reason of the fact than about its certainty, and the possibility of predicting it from conditions which do not already include it, or include it so that it was not formerly recognized.

In Kraepelin's book, we look in vain for the term secondary or terminal dementia. Since the collapse-delirium and the (rare) *amentia* (which comprehends only ½ per cent. of Kraepelin's cases) do not leave behind dementia, since the toxic psychoses are a topic by themselves, and of relatively good prognosis, and epilepsy and senility are anyhow distinct entities; moreover since manic-depressive insanity has one of its fundamental *raisons d'être* in the exemption from deterioration—we must see in the cases of dementia præcox the almost exclusive ground for the development of "secondary dementia."

Let us see what Kraepelin's distinctions are:

In *manic-depressive insanity* we found our diagnosis on a triad of symptom-pairs—exaltation and depression of feelings (as we have learned from Wernicke; not to be mixed up loosely with perplexity), flight of ideas or inhibition of thought, and irresistible psychomotor unrest or inhibition. These fundamental symptoms decide the diagnosis, and our final question will be: Are they traits of constitution and temporary exaggeration of individual make-up, or are they the expression of a special disease? To what extent are they at the core of the pathological process, and to what extent collateral evidence or individual response? Do they explain the tendency to recurrence and the absence of deterioration in these types?

And what are the fundamental traits claimed for *dementia præcox* apart from termination in a peculiar kind of mental weakness?

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[1] And to those who do not live in goiter regions it must be especially welcome to see a paradigm withdrawn with which they have only theoretical experience.

Kraepelin's description is not very direct. It follows his general plan of examination, which makes a dissection of one's experience with the patients, which may be distracting for one not hardened and trained to it.

The comprehension of external stimuli is usually not deeply interfered with; the patients often have a remarkably good understanding of what is going on even during great excitement, and their *orientation* as to place, person, and dates is usually surprisingly intact, except occasionally in stupor and profound anxiety. The correct perception is, however, at times concealed by delusional interpretations, and especially obscured by the frequent disinclination to give an account and the tendency to trifling or to intentional deception. *Hallucinations* hardly ever are missed in an acute or a subacute development of the disease. They either persist or fade slowly; at first they are usually unpleasant, and later they are taken with indifference. Those of hearing are most prominent, next those of vision and touch and sensations of currents of contacts and of various influences. They are frequently looked upon as artificial, as a kind of theater, sometimes amusing; it is usually difficult to get descriptions. Often they are absurd and incoherent, even when the patient is connected.

The sensorium or *consciousness* is perfectly and permanently clear in many cases. Temporary cloudiness in excitement and stupor is usually slight and often only apparent. *Attention*, on the other hand, is always deeply affected, owing to great distractibility, and, above all, owing to a lack of spontaneous interest, a lack of real desire to digest and understand. In deep stupor or advanced dementia it may become quite impossible to rouse any attention; on the other hand, there may be a stealthy curiosity, breaking at times through the negativism.

Memory is relatively little disturbed, surprisingly so for *fragmentary* knowledge of dates, school-information, etc. Also the acquisition of new numbers or names by apparently totally apathetic patients and their retention for days and weeks is sometimes surprising. But it is frequently difficult to get at the facts owing to negativism.

The *course of thought* always suffers profoundly sooner or later. Even apart from the incoherence in states of excitement and from stupor, there is, as a rule, an increasing *dilapidation of thought*. At first it may show merely in increased distractibility, sudden, erratic, often senseless changes of topic, and in the interpolation of superfluous phrases and digressions. This not infrequently culminates in a confusion of speech with a loss of all connection and with new words, often in striking contrast to the connected behavior and the real train of thought, in which the patient may grasp and even elaborate in good order, although even in this respect signs of stereotypy, clinging to single concepts, and the recurrence of the same limited phrases for weeks or months, show a reduction. Frequently there is also a tendency to rhymes, to senseless repetitions of sounds, to forced word-plays.

Without exception, there is also a profound damage to *judgment*. With all the retention of old knowledge and, in some ways, of its use, the defect of attention does not fail to prevent a real grasp on the situation; the most natural conclusions fail to be drawn; discrepancies and objections are not thought of. Hence the erroneous views on their situation and condition, the inability to grasp the seriousness of the disorder and of its consequences.

Extremely often transitory or lasting *delusions* form on this ground, at first sad, hypochondriacal, self-accusatory, or persecutory, later more expansive, but mostly contrary to all sense or at least more or less erratic and somewhat changeable. Even in the clearly paranoia-like forms the delusions gradually lose their force and connection, while in the other forms they finally reappear only from time to time.

Very striking and profound disorders regularly take place in the *emotional* life. The sadness, anxiety, or excitement of the onset or the rarer gaiety with immoderate laughter pass into more or less emotional dullness. This is perhaps at the root of the general indifference of attention, and not infrequently the first sign for parents and friends; matters of interest, ties of affection, even wonted pleasure (except perhaps the attractions of food), are neglected; and occasional irritability shows. The *demeanor and activity* seem to show a general reduction of voluntary impulses in general, quite plainly in the terminal stages, but often at the outset. The patient becomes lazy and neglectful; if excitement occurs it lacks definite aims, and is a planless discharge of inner tension. It is often an impulsive reaction to a sudden whim, without explanatory reasons, or at least no convincing ones.

The same inadequacy of control shows also in stupor, which is generally governed by the phenomenon of obstruction of will, a wiping out of every impulse by a stronger one in the opposite direction; this is especially manifest as *negativism* which is shown in resistance, aversion, direct perversion, or mere inaccessibility. Even in this stage sudden senseless outbreaks are observed, perhaps followed as suddenly by a relapse into stupor. Not infrequently the same impulse reappears, *stereotypies of motion and attitude* develop, so characteristic of catatonia, also verbigeration, mannerisms and oddities in many ordinary actions, which may persist for years.

Probably in some connection with severe damage to the will and to spontaneity, we further find *automatic responses to suggestion.* The patients become members of a herd, and in many of them there is temporary or permanent *catalepsy*, and often echolalia and echopraxia. Although these symptoms vary considerably there are probably few cases of dementia præcox who would not present the one or the other of these signs at some period of the course of the disease.

The *capacity for work* suffers very considerably without exception. The patient needs urging everywhere and is incapable of adaptation to new conditions. Work of a monotonous kind is often done faultlessly, but also thoughtlessly or with odd admixtures.

The *physical condition* is equally marked. Fainting spells and epileptiform convulsions, more rarely spasms of isolated muscle-groups (face, arm), tetany, or apoplectiform attacks and even prolonged palsy are noted as preceding the disease. At times such an attack of coma is the beginning. Kraepelin noted such incidents in 18 per cent. of his cases, excluding those cases of spasms or fainting spells in childhood the bearing of which is quite uncertain. Moreover, there were in several instances hysteriform spasms and palsies, aphonia, singultus, sudden rigidity, local contractures, etc., and lasting peculiar choreatic motions (athetoid ataxia). In two cases there were plain aphasic disorders during a state of dull cloudiness (inability to recognize and name objects for several hours although the patients could speak and made evident efforts).

The tendon reflexes are always increased, also frequently the mechanical irritability of muscles and nerves; the pupils are often wide or different; vasomotor disorders, cyanosis, circumscribed edema, dermatographia, and at times excessive perspiration are found. Saliva is often abundant. The heart's action is subject to great variation and is often weak and irregular. The temperature is usually low, the menses irregular or absent. The observations of disorder of the thyroid have probably only local interest. Anemia is very frequent. Once there was glycosuria, once polyuria. Sleep is much deranged; the appetite often perverse. The weight is very irregular, and is apt to rise finally.

Since the forms are very variable, a division into hebephrenic, catatonic, and paranoic forms is artificial and claims only didactic value. For a special description we must refer to the text. For our present purposes it may be sufficient to state that the paranoid forms are given the poorest prognosis; the hebephrenic forms show in 75 per cent. a profound dementia, in 17 per cent. a moderate dementia, and in 8 per cent. what might practically be called a recovery. Catatonia results in 59 per cent. in a peculiar and considerable dementia; in 27 per cent. the dementia is moderate or slight, and in 13 per cent. there are no residuals or but slight ones. A certain number of these are, however, likely to experience fatal relapses. Twenty per cent. of the patients show prolonged remissions resembling recoveries and lasting up to "five to ten years." The remarks on the reasons for the difference of prognosis (vol. II, p. 180) are of decided importance to us.

"To my regret I have not been able so far to find any definite points from which to draw conclusions concerning the probable termination of the individual case. Considering the recoveries, as it were, as remissions of long duration, we should have to answer the question: Which cases justify hopes for the occurrence of prolonged remissions, and after how long a time may we still hope for one? There is no doubt but that in general the attacks of rapid onset are more likely to lead to remissions than the slowly developing ones, very much as in general paralysis. Since an acute onset with vivid excitement is incomparably more frequent in catatonia than in the hebephrenic forms, we may well make this accountable for their more favorable prognosis. Moreover, the probability of considerable improvement becomes less in a measure as those peculiarities develop which we find so prominent in the large number of certainly incurable cases. We mention the loss of emotional responsiveness while comprehension is preserved; further, the fixed mannerisms and stereotypies, and finally the periodic unaccountable depressions and excitements. It is naturally merely a supposition so far, that the development of these and probably of many other signs means the approach of an incurable terminal state, yet many experiences are in favor of this. A very large number of observations, carefully planned and continued with this in view, will gradually make this matter clear.

"It is, of course, not always easy to be quite sure concerning the existence of those signs. Indifference toward the happenings in the surroundings may also be pretended, through negativism or through stupor. Only when the patients show absolutely no interest in their fellow-patients, their relatives and their occupation, notwithstanding complete composure and without the signs of negativism, may we conclude that the emotional responsiveness is actually annihilated. In a similar way, only the truly lasting and completely fixed stereotypies can be used to judge of the chances of recovery, and only those depressions and excitements which enlarge and disappear again suddenly after quite a short duration and at more or less regular intervals. On the other hand, the persistence of a decided negativism with stupor would seem to be open to far-reaching improvement even after a very long time. In definitely incurable cases the negativism usually subsides gradually; yet we know cases in which a negativistic stupor terminated in a surprising recovery with defect after three, five, and even eight years. Whether such cases imply probabilities or merely remote possibilities requires further investigation."

These passages should be sufficient to show the reader the caution of Kraepelin and the restriction which he put upon himself, and also the points which he is led to consider most fundamental for the working of the disease-principle which brings about the various results.

The last group created by Kraepelin and to be drawn into the scope of our investigation is that of *melancholia.* By this term are meant all the morbid anxious depressions of advanced age such as do not represent phases of other forms of insanity. Besides depression, there are always delusion formations, especially of sinfulness, but also of persecution and hypochondriasis. The onset is gradual; the patient feels poorly, is anxious and dull, worries, but has good and bad days. All doubtful actions ever done assume immense proportions; against the remorse no reasoning avails, and the ideas seem to be more than explanations for the depression. Fear of punishment and of the consequences of thoughts and actions add further elaborations. Hypochondriacal ideas of effects of ill-practices or interpretations of temporary sensations are fantastically worked out. Isolated hallucinations, scolding, or suggesting things, or visions of all sorts of absurd things, add to the torment. Sensorium and orientation remain clear, notwithstanding the occasional delusional statements that things and people are "different"; the stream of thought is coherent though somewhat monotonous, although the patient may complain of "so many thoughts."

A second group, of usually older patients, may be made of those cases who have more prominent erratic and absurd delusions and elaborations of what they see and feel; forms of "*depressiver Wahnsinn.*" Nihilistic ideas may govern the field, or at times meager delusions of an exalted character; hallucinations are more marked; the sensorium and orientation are frequently less clear. The stream of thought is confused and monotonous, especially during agitation; invariably anxiety is associated with the depression; but there is a certain absorption restricting the scope of interest. Relatives are apt to make a deeper impression on them than even very excited fellow-patients. Concerning their attitude, there are all grades of transitions of agitation. The course of the disease always shows a more or less regular curve. The prognosis is, on the whole, doubtful. Only 32 per cent. recovered, 23 per cent. improved enough to go home and partially resume their occupation, 26 per cent. remained uncured, and 19 per cent. died during the first two years. Among the cases below fifty-five years of age, 40 per cent. recovered; of the older ones, only 25 per cent. The unfavorable turn usually shows in a decrease of emotional excitement without a decrease of the morbid concepts, or with a development of absurd delusions. When depression and delusions finally fade, the patient remains dull and indifferent, easily discouraged and crying; in graver defect the patient becomes confused, forgetful, devoid of emotions; they stand around dull and lamenting monotonously. Others become inaccessible, childish, are in an attitude of self-defense, gesticulate and mumble to themselves. The loss of weight usually continues. Autopsies merely showed extensive arteriosclerosis, and in two older cases signs of beginning cerebral atrophy.

Sixty per cent. of the cases are women; most of the attacks occur between fifty and sixty. Etiologically, heredity is moderately prominent (frequently arteriosclerotic disorders); constitutional oddness and external condition seem to play a great part.

Kraepelin's description includes most cases of anxious malancholias, depressive "*Wahnsinn*," and the senile depressions. It ascribes all the depressions of younger years to manic-depressive insanity or dementia præcox, a few to degenerative insanity and perhaps also to hysteria. Manic-depressive insanity which may begin in the period of involution shows a more rapid and favorable course, signs of manic traits, such as considerable impulse to activity, flight of ideas, exalted ideas, cheerfulness, without any signs of dementia; in the actual depression, the behavior of a case of melancholia forms throughout the natural expression of their anxious or irritable mood; whereas in manic-depressive cases, indecision, retardation, and inhibition of all voluntary actions stands in the foreground. On the other hand, whenever they do show irritability, it is always accompanied by vivid response in conduct and utterances, whereas the irritability of the melancholic bears the stamp of inward anxiety. Recurrences are observed in 15 per cent. In some of the cases it seems that a previous attack occurred in the fourth decade.

Transitions to senile confusion are quite gradual. A differential diagnosis from dementia præcox will come into question, especially where catatonic signs of rigid negativism, peculiar silly excitements, and mannerisms develop, especially where the patient is otherwise composed and oriented. The differential diagnosis from general paralysis is said to be most difficult.

About a dozen cases that Kraepelin has seen within ten years, induce him to describe separately a presenile state of gradual development of great weakness of judgment with delusion formation and increased emotional irritability. Many hypochondriacal traits remind one of hysteria; the delusions are fleeting; they lack the connection and elaboration of paranoia, and the patient is quite inconsistent. Women are attacked in the forties or fifties, men a little later. Heredity is almost constant. The exclusion of dementia præcox is made probable by the absence of catatonic signs; what may resemble them is not simply impulsive, but always founded on delusions. The termination is never profound dementia, but a moderate weakness with a few changeable and disconnected delusions.

We now turn to the works which take a different standpoint.

The older writers on mania and on melancholia were obviously in as decided a position as the one taken by Kraepelin, when they grouped together the recoverable and the deteriorating first or second attacks as manias and melancholias. They considered it impossible to make a distinction according to the outcome clearly from the start. On the other hand, they pretend that the attacks of periodic mania are distinguishable from the simple non-recurrent attacks; at least they give rules of distinction and they make a great case of the distinction of excitement and depression.

On the European continent *the acute psychoses* have long been divided into more specific groups. Meynert's amentia and the types of *Wahnsinn* withdraw a large number of those cases in which a true depression or a true exaltation could not be called the chief and leading feature. This purified the groups of mania and of melancholia. Mania as described by Mendel (1881) is not essentially different from what Ziehen and the earlier editions of Kraepelin implied by it. Since our whole discussion would lack a safe foundation if I should take for granted the reader's knowledge of the subdivisions, I give a summary of Ziehen's groupings, referring, however, to Schüle's "Psychiatrie" as the best exponent of the older standpoint which governed the dissolution of the huge groups of what is called mania and melancholia in Anglo-American psychiatric literature.

Ziehen also adheres to the restricted types. From his first edition he requires for the diagnosis of *mania*—(1) exhilaration; and (2) acceleration of the association of ideas, and as part of the latter, motor agitation. All the symptoms must be derived from this formula. This leads, indeed, to a picture identical with Kraepelin's pure manic phase. Ninety per cent. are said to recover, 5 per cent. die of intercurrent diseases; according to the first edition, in 10 per cent. (probably misprint?), according to the second edition, in 6 per cent., there is merely a "recovery with defect" or a real secondary dementia. This termination seems to occur usually in "patients who have had several previous attacks and finally succumb to a severe one." (A publication of these cases would be very valuable.) Very rarely a case passes into secondary paranoia, or into chronic mania with persistent hilarity, flight of ideas, and motor restlessness without any intellectual defect. Even including the periodic forms, mania is a rare disease—only three or four per cent. of the admissions. A safe distinction from periodic mania and circular insanity does not exist. Periodic mania is probable where we see:

1. The depressive initial stage very little developed.
2. The rise and the decrease of exaltation very sudden.
3. Relation to menstruation.
4. Very marked heredity.
5. Hypomania with reasoning traits.

Circular insanity is probable where the initial depression is long and where depressions lasting hours or days interrupt the manic picture. A diagnosis is only possible after more than one double attack.

Ziehen distinguishes irregular recurrences from the periodic form (especially frequent with hypomania).

Melancholia, according to Ziehen, is similarly limited to those cases which develop and run their course with a morbid primary depression (with a decided true affect of sadness), and a primary inhibition of thought. The effect on the motor sphere is also that of inhibition; but this effect, as that of unrest in mania, is a consequence of the general disorder of cortical associations. He describes the simple melancholic depression, and the graver forms, and includes, further, the cases with anxiety, even if anxiety is the most prominent and even initial symptom. Recovery is complete in 90 per cent. of the cases. After long duration there may remain a certain difficulty for complicated topics. In but few cases silly laughter ushers in a secondary dementia, with increasing number of hallucinations and defects of memory and judgment. Or the melancholia lasts and becomes chronic. Secondary paranoia is rare. There is, however, a great tendency to recurrences, sometimes after a score of years or more (such as one attack at puberty, and one in the climacteric period); or to real periodicity. Hallucinatory melancholia is not as favorable and there are all grades of transition to hallucinatory paranoia. Or melancholia may be part of circular insanity. In the latter, Ziehen claims the occurrence of secondary dementia in 10 per cent. As to frequency, circular insanity precedes all other psychoses (?).

Stupidity, a disorder characterized almost altogether by inhibition of all association of ideas with practically no affect, is credited with 60 per cent. of recoveries. Recovery with defect, rather more rarely pronounced secondary dementia, or occasionally secondary paranoia, or death by intercurrent disease, or permanent apathy, make up the other 40 per cent. It is said to be an exhaustion disease of early years and rare after the thirty-fifth year. How often recurrences are seen is not said.

A large group of what in Anglo-Saxon statistics is customarily classed as mania and melancholia, is put under the heading of paranoia, *i. e.*, disorders on delusional or hallucinatory basis. Here only the acute forms interest us. The *acute simple paranoia* without hallucinations is always recovered from, but has a great tendency to recurrence. Numerous and changeable delusions come up, with variable affect; there is a secondary acceleration of ideation and agitation; orientation may suffer. The duration is rarely more than three weeks. The frequency is not mentioned. Far more frequent is the *acute hallucinatory paranoia* which Ziehen identifies with Meynert's amentia, or Fürstner's confusional insanity, which shows all stages between pseudo-stupor and great agitation. Nearly 70 per cent. recover (usually within less than one year), but many of these show recurrences in one, two, or three years, and very often the second, third, or fourth recurrence leads to secondary dementia or chronic hallucinatory paranoia. Scarcely 30 per cent. remain permanently well. Secondary dementia follows the first attack in at least 15 per cent. of all the cases, especially in young persons; usually the patients begin to give silly replies also outside of their hallucinatory agitation. The parallelism between affect and delusions is gradually lost, etc. Chronic hallucinatory paranoia may come about through cumulation of many recurrences, or following a single attack; delusions preponderate over hallucinations, and the auditory hallucinations over the visual ones. Death is very frequent in those forms which are known as *delirium acutum*. (Ziehen also classifies delirium tremens as a peracute hallucinatory paranoia, "which occurs exclusively in chronic alcoholism.") Ziehen also speaks of periodic acute hallucinatory paranoia (periodic amentia), with unfavorable prognosis; and of the much rarer periodic acute simple non-hallucinatory paranoia; and finally of *circular paranoia* (the type of acute hallucinatory paranoia with flight of ideas frequently passes into a stuporous phase and the cycle may recur). Recoveries are rare; but the intervals may grow longer. Finally he classes those forms of periodic insanity in which the types vary irregularly (mania, melancholia, and various forms of acute hallucinatory paranoia), as polymorphous periodic insanity. In a foot-note he adds: "The attempt of Kraepelin to combine all the periodic and circular psychoses and simple mania (and in part also melancholia) into one disease group, as 'manic-depressive insanity,' comes from a confusion of tendency to recurrence with periodicity."

On reviewing this brief summary (to do Ziehen justice one ought to read his descriptions, of which only a bare skeleton is given here, sufficient to hold forth the important issues of discussion), one sees in most forms the admission of "innumerable types of transition," and the division is recommended on account of psychological clearness. Ziehen does not state how many cases are of doubtful classification; nor does he attempt especially to single out the types of poor prognosis as far as lasting defects go. Acute simple paranoia has an absolutely good prognosis, next comes mania and melancholia with 90 per cent., and finally acute hallucinatory paranoia (which includes forms with or without disorientation, and only excludes the symptomatic deliria, and the psychoses of epilepsy, hysteria, etc., from what is often called "delirious mania" in America), with 70 per cent. and only 40 per cent. without recurrence in ten years, or 30 per cent without any recurrence. Just this last statement might well be taken *cum grano salis*. We shall also see the great importance of investigating and grouping the cases as to whether the attack shows a sufficient or a trivial constellation of etiological factors, a point not sufficiently used by Ziehen. Stupidity stands last among the acute psychoses, as recoverable only in 60 per cent. To complete the list of what is commonly classed as acute insanity in this country, we must include Ziehen's *states of clouded consciousness*. He separates them from acute and peracute hallucinatory paranoia, because of their sudden onset and termination, their short duration (minutes, hours, or days), the almost constant defects of memory (amnesia) for the condition, "such as hardly ever occurs in acute hallucinatory paranoia, including delirium tremens," and finally because orientation and coherence of the association of ideas are always interfered with, while hallucinations and delusions may be absent in exceptional cases. He admits that there is no sharp limit. He finds these states related to sleep and somnambulism, to hypnotism, to epilepsy (with numerous types), to hysteria, and finally on toxic, congestive, and angiospastic ground, or connected with migraine, neuralgias, affects, and traumatisms. Finally, he treats the symptomatic deliria (1, infective or febrile; 2, toxic; 3, on ground of defervescence or collapse; 4, on ground of inanition), the deliria of seclusion in the dark and the disorders on compulsive ideas.

Passing to Wernicke, we are put on totally different ground. No longer the skeleton of a psychology which could as well be derived from permutations of concepts of traditional psychology (affect, ideas, hallucinations and delusions, and trend of association, and memory), but—simple psychiatric observation? Yes, to a large extent, though characteristically stamped by a special terminology, only in part derived from the very excellent and faithful method of observation and description which we have praised in Wernicke. We continue our analysis interrupted in the earlier part of this chapter.

To start with, we find Wernicke's *anxiety psychosis* separated from melancholia, as with Kraepelin, who, however, preserves the name melancholia for just these forms and classes them as disorders of involution, as largely occurring in the climacteric or post-climacteric period. We, therefore, need not consider it in our correlation with manic-depressive insanity. It is sufficient to say that Wernicke sees in his anxiety psychosis anxiety as the fundamental trait, the source of morbid concepts, which are autopsychic in slight anxiety, allopsychic and somatopsychic in deeper forms. The ideas will often appear as phonemata (voices), and the other senses may hallucinate during the height of the affect, in epileptics even in the form of combined hallucination. The self-appreciation of the patient shows always depressive traits, while allopsychic orientation remains intact, or is only disturbed by irradiation of the perplexity and ideas of reference. The deductions from self-accusations lead to fear of ruin even of the family, and to fear of danger through others. Alcoholism, epilepsy, the climacteric period, and senescence furnish the ground for this form. The allied somatopsychoses are probably to be grouped with the paranoic psychoses. Wernicke admits, however, a certain group *of transition cases between anxiety psychosis and affective melancholia* (see later), especially in young people and in senility, and always with a good prognosis. The depression is more even, anxiety is subordinate; allopsychic ideas are subordinate. Further, certain forms of melancholia agitata may consist at times of a mixture of anxiety psychosis and the manic symptoms of flight of ideas and flow of words. The *acute hallucinosis* (simply hallucinatory insanity without disorientation) comes under consideration, inasmuch as not all, though most, of the cases are of alcoholic origin, and because it might be mixed up with anxiety psychosis. It is, however, more essentially hallucinatory, more continuous (much more independent in its course from the degree of anxiety as such). As acute expansive autopsychosis Wernicke presents (p. 324) a case of acute delusional insanity, not unlike a beginning of general paralysis—a man of thirty-eight, who had an attack of nervous excitement at seventeen, and a young man of twenty-two with a peculiar attack on a basis of autochthonous ideas with one short intermission, not purely "delusional," but apparently with a rush of ideas.

The range of the disease-groups which is quite certainly covered with Kraepelin's manic-depressive insanity is taken up on page 337. Wernicke presents two cases showing a diametrically different picture, one of *affective melancholia*, and one of *mania*, instances of pure depression and pure exaltation, and associated with them intrapsychic afunction or hypofunction, and intrapsychic hyperfunction. From the point of view of contents of consciousness, "autopsychic orientation" suffers first in the form of self-depreciation or exaltation (megalomania).

First comes *melancholia* with its intrapsychic afunction, which shows at once in the difficulty of decision, and the dread of the future and the feeling of subjective insufficiency. There is a dulling of interest and psychic feeling, a feeling of emptiness of life, self-accusation over the "neglect," a feeling of unhappiness (a special form of autopsychic perplexity, whereas hypochondriacal depression is a somatopsychic perplexity). Then objective symptoms develop: an inactivity (intrapsychic akinesis) in speech and action. An actual retardation due to great difficulty of reaction goes beyond the simple picture called *affective melancholia* by Wernicke. He ascribes to "*depressive melancholia*" (which had better be called hypokinetic melancholia or, even better, hypokinesis; and which is probably [see p. 443] a product of deduction from the general schema) initiative mutism and initiative akinesis; also diminution of expressive motions, but less of simple reactive motions; the affect of subjective insufficiency also vanishes; instead of dominant thoughts, there is a dearth of thoughts; the fear of bodily danger might still allow of anxiety; but there is no flexibilitas, pseudo-flexibilitas, rigidity, or negativism. *Affective melancholia* also may show anxiety as an immediate consequence of the intrapsychic inhibition, and on the same grounds, dominant ideas of a hypochondriacal nature. Moreover, there are disorders of appetite, cool extremities, etc. In *severer types* of affective melancholia, an irradiation may lead to fantastic depressive delusions—so many people are sick, the world will come to an end; the patient is not human, can never die. It may also lead to refusal of food, to isolated visions, or to phonemata. The course is; as a rule, continuous; the attack is often followed by a short manic condition. Very few cases become chronic. Even in this very narrow limitation affective melancholia is a very frequent psychosis. It is easily distinguished from the anxious psychosis (which has more fluctuations, little feeling of subjective insufficiency, and more allopsychical elements, and not even an indication of retardation). The distinction from hysterical and neurasthenic states of anxiety is somewhat less easy, especially where the latter have a very pronounced impulsive desire for suicide. Depressive autopsychoses show circumscribed autopsychic delusions of reference of the nature of dominant ideas (self-reproach over the death of a relative), or of compulsive ideas, and are a picture distinct from affective melancholia.

This brief sketch will show, I think, how painstaking and true to facts Wernicke is in his discriminations in this one large complex of cases which are all juggled together as melancholia in our statistics. Wernicke claims for the small well-outlined group of affective melancholia a perfectly good prognosis, even in advanced age, although there the chances of recurrence become greater. He recognizes the relationship with those depressions which Kraepelin describes in manic-depressive insanity, but denies that recurrence is the rule, except where the attacks set in and end abruptly. He fails, however, to give an exact statement of his actual experience. *Other depressions*, not in the frame given here, must be analyzed and classed as different. It would seem, though, that the limit of affective melancholia and depressive melancholia is not as pronounced and not as essential as Wernicke makes out. As especially pleasing to me, I refer once more to the simple statement that affective melancholia consists in depression plus intrapsychic akinesis, instead of what Ziehen and Kraepelin speak of as "*Hemmung*," or inhibition, a term too easily used and apt to suggest a *deus ex machina*. The simple description—akinesis, or retardation—is decidedly more objective.

On his instance of *simple mania*, Wernicke describes first the flight of ideas, in which the accidental fails to be suppressed (attention is not normally held to a topic except with an effort, or not even then). The purely affective simple mania, however, does not reach a degree of *decided* incoherence. The morbid euphoria leads to autopsychic disorientation in the form of exalted ideas and delusions, which may even become fantastic, but change easily and are more whims than convictions. The increased activity of association leads, further, to a certain degree of distractibility (totally different, however, from true hypermetamorphosis), and to inconsiderate and hasty action and even irascibility. In addition to this, a veritable unrest leads to almost compulsive overactivity and tendency to useless pranks, and a great productivity in talk with puns and alliterations. Only when irascibility is prominent do we find temporary intermissions from talking. In it all there is a distinct leveling down of the normally dominant trend of thought, the very reverse of what happens in affective melancholia; egoism, lying, lack of modesty, and a total change of "character" develop. The rare and merely occasional phonemata seem to be largely expressions of ideas of exaltation. The general condition of the patient is good. The onset is usually rapid, the recovery a little slower. Apart from intercurrent risks, mania in this sense is the most recoverable mental disorder. It occurs like affective melancholia, chiefly on a basis of heredity and neurotic degeneracy.

Wherever *additional symptoms* occur they determine a *different diagnosis*. Wernicke mentions paralytic mania (the exaltation is one of conviction), the hyperkinetic traits, and, especially in the recurrent forms of mania, phases of confusion (depending on flight of ideas, and only in severer cases complicated by additional elements). Wernicke speaks of the great tendency to recurrence, of the peculiar relation to affective melancholia, of the spurious and veritable circular forms. The fact that he excludes from his picture what appears at the height of most attacks of mania, and what is bound to occur in the course of recurrences, shows how firmly Wernicke adheres to the symptom-complexes and how, in contrast to the broader clinical attitude of Kraepelin, he is not in the least concerned about the cases which do not fit his definitions. That he also admits a chronic mania, *i. e.*, a chronic persistent picture like the one described, although he is sure that it never develops from acute simple mania, would be another deviation from Kraepelin's standpoint, to whom general course and bearing mean more than symptomatic make-up of the disorder as such.

That which constitutes the majority of the ordinary manias in the international traditional meaning of the word is treated by Wernicke under *hyperkinetic motility psychosis* and *confused mania*. The term delirium does not appear, and Meynert's amentia is dealt with under "confused mania." This general group obviously contains, therefore, many cases which Ziehen classes as acute hallucinatory paranoia, since Wernicke's acute hallucinosis excludes those with disorientation as to time, place, and person.

For the sake of closer connection we shall first review Wernicke's lecture on *confused mania or agitated confusion*. Clinically (Wernicke himself uses this word here), it is an acute psychosis which may begin and end as a mania, but in its course presents, in addition to the symptoms of pure mania, psycho-sensory and psychomotor "disorders of identification." When the purely manic phases are very insignificant, we do well to speak of agitated confusion, in which the sensory or the motor agitation may be more prominent. Usually the sensory and the motor agitation are combined. When the motor agitation prevails, we find transitions to the hyperkinetic motility-psychoses. The confusion, as such, is not the decisive feature. We must distinguish whether the confusion is a symptom of irritation and allied to the productivity of speech, of flight of ideas, or whether it is a condition of defect, a simple incoherence from dissociation or sejunction. Only the former is to be considered here. Confused mania is limited to associative excitability, in which the attention of the patient is fleeting, but not so that it could not be fixed for moments; in which the incoherence is a formal disorder, a simple exaggeration of intrapsychic hyperfunction, a flight of ideas, in which similarities of words play a great rôle, and in which we may lose the appreciation of connection between the words and fragments of words produced. In this sense confused mania occurs as the acme of rare cases of simple mania; it is much more frequent in recurrent mania and especially in periodic mania, and most frequent in that of menstrual origin. The cases he mentions are splendid pictures of what is called mania by Kraepelin and by all others who limit the use of the term to the simple exaggeration of hypomania. Thus, he sums up a fifth attack of periodic puerperal mania in a patient (whose first attacks had had more the features of a hyperkinetic motility-psychosis); incoherent flight of ideas with occasional quick appropriate replies, an exuberant exalted attitude and correspondingly exaggerated expressive motions, impulsive motor activity expressed by tearing and swearing; immodest manners; in addition to this, an admixture of motor hyperkinesis, acting as a chansonette singer, and hypermetamorphosis. With this form, periodic mania is not exhausted. Wernicke singles out all cases with even temporary allopsychic disorientation (non-recognition of place, situation, and persons and objects), and deeper disorders, and calls them phases or attacks of periodic maniacal allopsychosis. Even very prominent flight of ideas need not produce disorientation; on the other hand, the *periodic maniacal allopsychosis* may become a "total sensory psychosis." The incoherence always affects first the autopsychic field, and only in the second and third place the allopsychic (appreciation of the outside world) and the somatopsychic field (the most fundamental consciousness of the somatic feelings). Besides the confusion through precipitation of activity, Wernicke describes, further, the *asthenic confusion*, either following very severe excitements of confused mania (a good description, by Wernicke, p. 405), or primary, perhaps totally independent of mania—a picture resembling much more closely Kraepelin's very narrow conception of amentia (a characteristic case is reported on p. 406). Meynert's amentia, according to Wernicke, would include chiefly his acute autopsychoses, allopsychoses, somatopsychoses, motility-psychoses, and their combinations, implying various degrees of *weakness* of association.

We now turn to the *motility-psychoses*. We have seen in a previous part of this chapter how Wernicke comes to this term. On reading his lectures, we find ourselves confronted with a vast material, exceedingly difficult to abstract, and obviously covering forms of very heterogeneous bearing for a comparison with Kraepelin's groupings.

Wernicke presents a patient of twenty-seven, who passed through a normal confinement; on her return from the lying-in hospital she showed abnormal anxiety about the child, and the second night began to sing and dance and talk of hearing angels sing. The next day she mistook her husband for the physician. For four weeks she had been in continuous motion, with mostly theatrical-pathetic expressive movements and a peculiar chanting accompanied by an odd rapid tremor of the lower jaw, unhappy facial expression, and refusal of food. This condition had passed into one of exhaustion, vacant staring, still exaggerated but greatly variable mimic play (she shows the white of her eyes, then frowns, or pouts or pushes forward her jaw), and occasionally oddity in pronunciation; whereas at other times she talks plainly, gives her name, the date of her confinement, complains of dizziness and fatigue, recites the Lord's prayer correctly, but adds an explanation to one of the ten commandments and begins a choral, quite correctly, but without being asked; at times she looks aside as if listening, or at the nurse as if for help; she has difficulty in directing her attention. She allows the arm to be elevated and holds it there a while; bending of the head is slightly painful. The mood varies between apathy, euphoria, irritation, and perplexity, and orientation is imperfect; there are even some somatic complaints (that she had been hunchbacked last night). Dancing and singing seemed to her as if forced on her. The patient is later demonstrated again in a state of catatonic stupor. From this picture Wernicke analyzes especially the hyperkinetic, parakinetic, and occasional akinetic traits.

On page 374 he gives another splendidly characteristic picture of an unmarried woman of thirty-six, who had a depression of three months at twenty-eight, and lately a brief premenstrual excitement, and two months later this typically hyperkinetic attack: The patient comes in dancing, singing a waltz-tune; five times she puts the flat hand on the head, says "holy water" and bows; she sits down on request, but gets up again, bends forward so as to throw her hair over her face, and repeats this twenty times; then she goes about, gesticulating and talking disconnectedly, with emphasis where her movements are emphatic. Her mood as well as the motions vary continually; at times they are overcheerful, at times exalted or irritable; only rarely does she give a correct reply; usually she ignores questions, while she is obviously easily guided in her motions by *casual* impressions. Her fragmentary talk is remarkably adapted to the temporary activities—there is, as it were, a *flight of activity* with associated flight of words. The whole performance might be called hysterical, but it continues when nobody is present, and after the attack the patient gives a clear description of the pseudo-spontaneous and inexplicably-forced nature of the movements. They have a certain monotony and even rhythm which may be accompanied by verbigeration; the motions are exaggerated, obviously unpremeditated, more adaptive, or directly forced. The patient sings, or goes through gymnastics, although she does not feel like it. Not infrequently there are phases of mutism. Almost always we observe hypermetamorphosis. *i. e.*, a compulsive distractibility of the trend of thought. When the latter is not much disconnected, there is nevertheless a tendency to false pathos, biblical oratory and an empty form of preaching. In two further cases Wernicke describes a remarkably pure picture of motor reaction to a peculiar motor uneasiness, "jactatoid (or, we might say, jactation like) motor unrest." Wernicke describes choreatic unrest, also certain types of impulsive actions (one case actually with one week's exacerbation of the nature of choreatic motor unrest), probably because these show no additional mental symptoms such as would be incompatible with the motility-psychosis. He, however, excludes motor reactions on a directly hallucinatory basis (p. 389); and ignores that he might well class some of his instances of forced motions as "autochthonous" motor attitudes or reactions (one instance of actual dementia præcox, another one with recovery), as not so essentially different from others with actually hallucinatory basis. Like the reactions on a hallucinatory basis, those in the motor unrest of perplexity have distinct marks of psychic motives; also those in delirium (tremens or paralytic). The same holds for the truly hypermetamorphic motor unrest (by irresistible distractibility, where the sight of a basin makes the patient wash himself, etc.). Just this form of irresistible distractibility occurs often enough in the hyperkinetic psychoses, and might well show Wernicke the danger of giving too much importance to a schematic derivation and classification of symptoms, and of tearing apart connections perhaps more natural than those of his system.

All these types may be covered by the vague expression of maniacal excitement, but might well be kept distinct as motility-psychosis, when no other symptoms occur. In this way we obtain a number of cases of pure hyperkinetic psychosis, while in other cases we merely find the symptom-complex as part of a wider picture. The pure cases are mostly irregularly remittent and form a large proportion of the periodic manias of the statistics, often of menstrual or puerperal origin, but also occurring in men. The attacks are usually short. The prognosis is generally good. Most cases recover after a number of periods, even when arising on a hereditary or "degenerative" basis.

Wernicke (page 392) explicitly states that *mania* is the only disease which shows actual transitions to the *hyperkinetic motility-psychosis*; and for this reason an inner relationship. On the other hand, the hyperkinetic motility-psychosis may be a phase of a cyclic motility-psychosis or an episode of a "*total motility-psychosis*," which we must take up here. It has probably much bearing on catatonia, which we can not treat fully here.

Absence of motility, even more than excess of motility, cuts us off from correct information of the mental life of the patient. Here we need all the more an accurate knowledge of what motor symptoms there are.

Wernicke gives first a demonstration of the first case of hyperkinetic motility-psychosis in the *akinetic* condition into which she had finally passed: a state of exhaustion, motionless and without response to calls. No fixation of the eyes, merely a slight increase of the frequency of winking. The arms show typical flexibilitas cerea, the hip-joints muscular rigidity. The legs are relaxed; the reflexes of the legs are normal, those of the arms increased; the head is freely movable. Only the jaws resist, and the eyelids, which had become closed in spasmodic weeping following pricks in the face. Pricks of the arms had had little effect, and the right hand had not been withdrawn at all; the toes of the foot went upward, and repetition of pricks of the sole made her bend the knee and knit the eyebrows. Pain evidently made her break through the reactive akinesis. Requests to move or to get up are not responded to or only very tentatively; when started and helped, she stood, followed like an automaton, but finally stood alone; when asked to walk, she slowly leaned over and, on the point of falling, suddenly started, ran as in the propulsion of paralysis agitans, and happened to drop on the couch. Throughout this test she had been sighing and weeping. The previous examination had shown disorientation in the three fields of consciousness.

The second case is one resembling hysteria, and is obviously lacking in detail, since no examination for parasitic ideas was made.

The third case is a delirium with episodic and later permanent akinesis, with temporary muscular rigidity; mutism interrupted by inarticulate outcries; relaxation after the eleventh day; utterances over peculiar sensations during the third week; improvement during the fourth, and an account of peculiar dream-episodes; the patient felt that he was a steamer, he had to put together the world, which had gone to pieces, etc. He remembers no reason for the rigidity. In the seventh week a slight relapse after an indiscretion (feeling that the brain moved in the skull, etc.), but complete and lasting recovery.

The fourth case, a young lawyer, Hebrew, began suddenly with anxious perplexity, attempts at suicide, mutism, negativism, odd attitudes. Death from pneumonia in a few days. Then follow cases showing queer attitudes, queer rope-dancer's gait, etc.; and the record of a young man of thirty-two who was suddenly taken ill and held his tongue squeezed between his jaws (although his eyes showed that he was conscious), the same position being held even after the jaws were separated in narcosis. He gained command over the tongue last of all when he regained his movements, on recovering from the sleep-like state in a few days. Complete recovery.

Wernicke gives an analysis of these symptoms of a motionless condition (akinesis), varying from apparent death to the catatonic stupor with mutism, negativism, catalepsy, rigidity, lack of spontaneity, parakinesis, verbigeration, stereotypies, and pseudo-flexibility (on suggestion); and he analyzes the depth of the sensorium and defines in detail the pure akinetic motility-psychosis, which is quite limited and excludes the types of intrapsychic origin of the motor state. The cyclic and complete motility-psychoses (with akinetic, parakinetic, and hyperkinetic symptoms) give an opportunity to discuss a few very peculiar pictures. Among the motility symptoms of psychic origin Wernicke discusses foremost his "depressive melancholia," which I should prefer to call hypokinetic melancholia, and he defines it as a part of a motility-psychosis, or as a pseudo-melancholia showing distinct paranoid traits, or as parts of general paralysis, or as the beginning of other complex psychoses (*i. e.*, very heterogeneous entities). In intrapsychic akinesis the disorder is said to be always even and general, without distinctions between the various muscle-groups (where would he class hysterical stupor with catalepsy of only one arm?). Speech is said not to be more involved than the other spheres; initiative movements are more affected than reactive movements, expressive movements scarce, but never totally missing; especially the eye will always follow the speaker (?); moreover, the specific motor symptoms of the simple motility-psychosis are absent.

Compulsive speech, syncopal states, hypochondriacal palsies (in limited muscle groups), psychical palsies, and fixed contractures are considered, and a discussion of the possible meaning and origin of the motility symptoms closes the chapter (which is rather vague and makes one conscious of the obscurity of this whole topic). Notwithstanding this strictly observing attitude and keen disquisition on the facts, one becomes conscious of the immense difficulties in the way of a thorough understanding of psychoses through the mere analysis of the nature of the symptoms. A great longing for simplicity begins to clamor in us, and who would not hail the very attractive structure held out to us by Kraepelin, if it were not for the fact that clinical experience seems to remonstrate against simplicity where things are actually very complex?

For a beginner, Wernicke's whole plan seems, at first sight, very difficult to grasp. Considering the small amount of analytical knowledge that is actually necessary to do one's duty in a hospital for the insane, such a burden of puzzles and analyses would to many appear overpowering and meaningless. Even for one who seriously tries to learn what there is, without any worry over the burden, the feeling is pardonable that there seem to be more names than can be made use of comfortably for the distinctions offered. After all, practical life is what confronts us. We are physicians to patients, and we have a right to say that the use of generalizations ought to have its vindication through the help they bring in specific instances. The merit of Wernicke lies in his faithful and accurate descriptions, and these place him above all reproach and question; and he certainly removes the blinding dogma of traditional nomenclature from a number of very important facts. But his system of analysis seems to totally blind him to very important issues which we might well begin to utilize.

The principal concepts Wernicke gives us are: *first* that of sejunction. Its utility is problematic, and it is a mere transcription of derangement of a habit, of adequate appreciation of things (by hallucinosis and false interpretations), of thought, reasoning, action (various types of delusions); *second*, that of the divisibility of the topics of psychic activity into three topographically outlined spheres. Both these concepts seek far to make the appearance of having something to do with ultimate cerebral localization, but neither with much hope of carrying conviction. The case is somewhat similar to that of some other concepts of general psychopathology, such as that of the *autointoxication* theory of so many writers. Purging and purifying the blood have so long been the doctor's chief reliance that the autointoxication theory naturally flatters the mind of those whose empirical habit is thus supplied with a scientific motive (to eliminate poisons even if there be no evidence of poison in many disorders in which a purge may do good). A gain in *localization* might similarly please the neurologically inclined physician, but even there the sober ones will ask: Where is the evidence? There are, however, many advantages in Wernicke's divisions, both as to method and as to results. What he arrives at with his theories appears, indeed, to have a good practical foundation; but the harmony between the deductions from his theories and the actual observations does not necessarily prove their correctness. However this be, Wernicke's descriptions and analyses have at least the merit of being the best we have to-day, and this is not to be forgotten by the critic.

Ziehen, who has conceived a definite psychology of his own, naturally turns to it for the purpose of teaching psychiatry, because of the love of the student for reconstruction of the complex reality out of a small number of elements which can be remembered a tolerable length of time. Wernicke creates his psychology with the needs of the neurologist and localizer in mind. But he does more. The standard emotions of exaltation and sadness are correctly supplemented by those of anxiety, perplexity, and irascibility; the mechanisms for adequate psychic adaptation or plasticity are also very justly divided. Those concerning the outside world, and our relations to it, are open to many illusions and false interpretation even in ordinary life, and this is easily increased, by defective states of the sense organs and by defect in our critical elaboration (allopsychic disorientation), into deliria. Those concerning our own personality, character, etc., are more apt to be disturbed from other sources; they are very delicate and their derangement of tremendous importance for our entire conduct (autopsychic disorientation). Those concerning our body are most automatic, and least open to correction by outside criticism, and to a large extent resting on unconscious habits; and their disturbance (somatopsychic disorientation) is apt to be a sign of rather grave importance. It further becomes quite obvious that many functions are shown to be deranged in insanity which could hardly fit into traditional psychology, but are properly put together as preeminently motor. These points cover in the main Wernicke's attitude. The discrepancy begins where we see Wernicke anxious to claim a decided advantage for the close limitation of a combination of a few symptoms where the existence of transition-forms shows so plainly that there can be no profound natural difference. In the matter of relationship between mania and confused mania, Wernicke himself admits this. This admission in one place might have given him a hint as to the advantage of a broader view. Again, under the heading of akinetic motility-psychosis, conditions seemingly very distinct in nature are ranged side by side, where separation would have added greatly to our interest.

In this respect Kraepelin has undoubtedly shown a broader and more helpful attitude. The great question of the relatives of the patients and of the physician himself is undoubtedly, "What will be the fate of the patient?"

Kraepelin has put together what is most helpful for this question. Wernicke makes, however, some severe comments on the freedom which Kraepelin takes in shaping his dogma regardless of difficulties.

What do his contemporaries say concerning *dementia?*

We have seen that Ziehen records about 6 per cent. of mania and melancholia (in the narrow definition which he gives these forms) as terminating in secondary dementia. In acute hallucinatory paranoia (the large body of acute psychoses, according to Ziehen) at least 15 per cent. terminate this way at once, and this is brought into connection with the accounts of cell and fiber degeneration in these cases. Yet even Ziehen says in his second edition that the diagnosis of secondary dementia was made altogether too frequently formerly. "Careful observation has shown that in many cases which formerly were counted with secondary dementia the intellectual defect is not secondary, but accompanies the psychosis from the beginning. In this case it is not a secondary dementia after a functional psychosis, but a primary 'defect-psychosis,' simulating in the beginning a functional psychosis. Confusion with hebephrenic dementia is especially easy, for its initial stage often presents the picture of a melancholia, or mania, or hallucinatory paranoia. To determine whether an intellectual defect has existed from the start is often extremely difficult. One has to depend on the frequently unreliable history furnished by relatives, and moreover the testing of intelligence is made extremely difficult by coexisting disorders of affect. Without anamnesis or observation from the onset of the disease, the diagnosis is frequently quite impossible. One can only say that pronounced tendency to stereotypies and perseveration and to catatonic conditions is somewhat more frequent with dementia hebephrenica."

In his first edition he merely admitted "*hebephrene Modificationen*" of mania, melancholia, etc., and saw in the symptoms only a pathological caricature of certain traits which belong to normal puberty. Hebephrenic dementia did not exist. In the second edition "dementia hebephrenica or hebephrenia is a defect-psychosis which begins at puberty and is characterized by a progressive primary defect and several characteristic accompanying symptoms (apathy, stereotypy, inhibitions, etc.)." He describes a simple form, a paranoid variety, a catatonic variety, and a circular variety (with cycles of one to four week), and mentions the decided tendency to remissions resembling complete recovery which create the false impression that the actual hebephrenia has been preceded by another psychosis, such as melancholia. He finds these cases much more frequent in Holland than in Jena. He limits their occurrence to the period of puberty, and exceptionally in retarded puberty up to the twenty-fifth year, and states explicitly that those who claim to have seen cases of dementia præcox even in the sixth decade, probably confused it with dementia secundaria, dementia traumatica, etc.[1] His remarks on differential diagnosis are very interesting, too, for their strict adherence to the meaning of *words*, where others would have taken a larger view of the *facts*. *Catatonia* is maintained as a "rare disease" with four stages—a melancholic, a maniacal, a stuporous one, and one of secondary dementia. One of the first three stages may be absent. It may occur at any age. Recovery is rare (evidently here Ziehen does not accept Kahlbaum's views). Many cases described as such are instances of the catatonic form of chronic hallucinatory paranoia and of dementia hebephrenica. Ziehen has "seen only two cases in which the intellectual defect certainly appeared only secondarily, and which corresponded to the definition of Kahlbaum in course and symptoms, without any focal disease found at the autopsy." He mentions one perfectly typical case, in which, however, he found a partial old softening in the region of the arteria cerebri anterior, of possibly traumatic origin, and mentions the frequency of catatonic symptoms in general paralysis, and in senile, traumatic, and epileptic dementia, etc. In the first edition the catatonic stereotyped attitudes and movements are said to occur chiefly in melancholia attonita, paranoia hallucinatoria, and any form of dementia. Considering the fact that Ziehen was originally an assistant of Kahlbaum, it is remarkable that he does not see the purely theoretical origin of Kahlbaum's definition of four stages, in the Zeller-Neumann theory of insanity, and the absolute freedom with which Kahlbaum himself treated his definition. Kahlbaum found the prognosis favorable. Still, Ziehen adheres to the letter rather than to the meaning of his first master.

A more conventional position is taken in this matter by Mendel in his recent "Leitfaden der Psychiatrie" (1902). He still holds to the ordinary primary psychoses and secondary dementias: delirium hallucinatorium with 80 per cent. (in asylums only 45 per cent.) of recoveries, mania (no definite percentages given beyond the statement that 80 per cent. recover), melancholia (hypomelancholia always recovered from, while only 60 per cent. of the real melancholia recover); in circular insanity final dementia is the exception; in paranoia he admits a stadium dementiæ (without percentages), and in acute dementia recovery in 80 per cent. As a special form of hypochondriacal melancholia he mentions those cases which rapidly lead to dementia under the influence of puberty and constitute a large portion of the cases of hebephrenia and catatonia. A reference directs the reader to the chapter on etiology, where Kraepelin's dementia præcox is summarized as a "special course of such psychoses appearing during the period of puberty." Mendel states that 2 to

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[1] He does this without inquiring into the possibility that there are really cases at that age which are characteristic defect-psychoses from the start; and he forgets that before he admitted the "dementia hebephrenica" he made the same blunder which he now wants others to continue, simply because of his narrow adherence to the name "hebephrenic" as referring to age—the same trouble to which we presently refer concerning catatonia.

3 per cent. of the patients in Berlin belong to this class. Yet he does not give the group a special place in his "special psychiatry." Furthermore, in the general portion of his book, in the chapter on the outcome of psychoses, he gives a description of paranoia secundaria ("developing from delirium hallucinatorium, mania, or melancholia"), acquired dementia, agitated dementia or demented confusion (the outcome of melancholia, mania, or paranoia), and apathetic dementia. "The question why in one case a functional psychosis terminates in recovery, and in another in incurable dementia, can not be answered to-day. Experience shows that especially those functional psychoses which appear at puberty, and in which there is marked heredity, pass frequently and rather rapidly into dementia (dementia præcox, in which Kraepelin distinguishes a hebephrenic, catatonic, and paranoid form)."

Wernicke's standpoint forbids the recognition of a hebephrenia because he will not admit any types as identical which have not a uniform symptom-complex. We have seen that, even in general paralysis, he speaks of an "etiological group" of paralytic psychoses. We find, indeed, that he has given up Kahlbaum and Hecker's hebephrenia as a disease-type. Clinical experience has made him abandon it more and more, and to-day he can only stand for the very considerable importance of "hebephrenic etiology." He accepts Kahlbaum's "heboid"[1] as a specific form of puberty-psychosis only, a moral auto-psychosis, *i. e.*, a relatively rare type not accepted in any text-book except partially in Wernicke's.

In the lecture on acute autopsychoses he gives an account of so-called alternating personality chiefly on hysterical or epileptic or alcoholic degenerative foundation, and of those cases of reasoning mania, which, according to French authors, recur with always the same scheme and an often surprisingly uniform plan of action;[2] and of certain cases of periodic alcoholism.[3] This moral autopsychosis in its climacteric form is mostly of unfavorable prognosis, and in its senile form probably always a transition toward senile dementia, but in its hebephrenic form relatively curable. The instance he gives is that of a girl with transitory loss of all discipline, feeling of responsibility and modesty, a tendency to obscenity but total absence of impulsive talking, flight of ideas, and motor unrest. Within three years there followed three recurrences, and in the interval slight depressions. One might have thought of circular insanity, but that would not do, "if one desired to cling to the definition of it as a regular alternation between manic and melancholic states." The leveling down of the dominant moral concepts "with the not infrequently total absence of intellectual disorder" is indeed similar to what occurs in mania; but the other cardinal symptoms of mania are absent, and Wernicke is nowhere willing to admit the "*formes frustes.*" Kahlbaum's cases are certainly noteworthy, and I am able to point to several such cases of my own which had a far more undecided position between Kraepelin's manic-depressive insanity and his dementia præcox than Wernicke's instance.

In the typical "heboid" (the exaggeration of features of puberty) Wernicke adds to Kahlbaum's description the symptoms of onerous sensations, onerous ideas, and hypochondriacal sensations as constant accompaniments. We should refer to this group again in a discussion of "*formes frustes,*" or of *vague types.*

As rather characteristic of hebephrenic etiology Wernicke mentions "hebephrenic expansive autopsychosis," a "primary paranoia," which passes rapidly into attonity and dementia. Next in frequency he finds motility-psychoses, especially the akinetic form. Very characteristically he says that the well-known tendency of akinetic motility-psychoses to pass into dementia, as well as the erroneously claimed tendency to recurrences, might in part be referable to this etiological relation (*i. e.*, the origin during puberty). Yet he knows of cases of akinetic motility-psychoses during puberty which recovered completely and permanently. "Hence the unfavorable prognosis which Kraepelin gives in these cases does not even hold without exception for the cases during puberty." Another frequent picture during puberty is the hypochondriacal anxiety-psychosis, which may remain abortive, or develop further, and, perhaps after an intercurrent phase of "total sensory psychosis" (= delirium), with a trend of fantastic hypochondriacal persecution, pass into a very characteristic hebephrenic dementia such as Kahlbaum describes. Certain acute attacks of "weakness of association with autopsychic and allopsychic defects, but without any signs of irritation on the motor side," in young girls, recovered after a prolonged paranoic state. Considering the relative difficulty in getting their attention, they have a surprising grasp on what they observed. These cases, too, although recoverable, seem to be on a hebephrenic basis.

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[1] "Allg. Zeitschrift für Psychiatrie," vol. xlviii.

[2] An illustration is given of a clerk who periodically assumed the rôle of a physician and committed apparently shrewd embezzlements, but always in the same way; this patient finally ended in epileptic dementia. The term reasoning mania and the term moral insanity have gradually lost the meaning they originally had, and they are incorrectly applied to what the meaning of the word seems to imply to the modern mind, unless one return to the old sources; a good illustration of the power of word over sense of accuracy.

[3] The "pathological intoxication," however, is classed with the true dream-states, such as most of the pre- and post-epileptic psychoses. (I mention this to give another instance of Wernicke's respect for the symptom-complex notwithstanding the fact that the essential cause or condition is the same for several types of disorders.) Wernicke describes a type of so-called acquired moral insanity (in the modern sense of the word; see an account of its history in my article in Baldwin's "Dictionary of Psychology," vol. ii).

I mention these with some detail because we need the facts in our comparison with Kraepelin's step in advance. Wernicke does not say (any more than do Ziehen or Mendel) just in what way *puberty* is accountable as a factor in etiology. He attacks "hebephrenia" in its original sense (as a feature of puberty), whereas *dementia præcox* in Kraepelin's sense refers more to the fact of a special form of deterioration and not so much to the age of onset. Wernicke sees the characteristic feature of hebephrenic etiology, not in the childish manner and silly ways that appear quite natural in dementia at this period of life, but in the tendency to unfavorable outcome (or, where they end in recovery, in the tendency to temporary symptoms of defect). He sees other characteristics in the predilection for certain disease-pictures, which, however, do not belong exclusively to the period of puberty; in a tendency to recurrences, and where they have a chronic course, to exacerbations, so that the most acute attacks occur as intercurrent episodes. Moreover, most of the acute cases, and in part also those of chronic development, take their course as compound psychoses, *i. e.*, at successive periods they produce totally different disease-pictures. He refers to a case which he demonstrated first as an affective melancholia, in the following semester as hyperkinetic motility-psychosis, and finally as a condition of pure defect in deep dementia, and he describes how vivid the face looked in contrast to the emptiness of mind, in distinction to what happens in general paralysis. This whole case is an instance in point of the difficulty of that "prognostic diagnosis" which Kraepelin makes so attractive an aim of psychiatry.

To complete this review, I must refer to Wernicke, page 544, where the forms of acquired dementia are treated connectedly. After a brief description of its effects of traumatism (in which he admits the possibility of a progressive degenerative process started by the trauma), of the mental aspect of multiple sclerosis, of lues cerebri, of meningitis, he says: "At the period of puberty one sometimes observes a dementia which may develop relatively rapidly and which may become very deep without being accompanied by essential psychotic symptoms of another kind. In other cases such a primary degenerative dementia, preceded by attacks of severe headache, develops at puberty, with delusions of reference of a harmless character, especially autopsychic delusions of reference (what the patient sees or hears is interpreted as having some reference to his thoughts; or hallucinations are interpreted in this way; the patient has the feeling that his thoughts are spoken aloud, etc.). Cases of recovered primary dementia, subacute in development, and outside of the period of puberty have come to my notice several times; but I should prefer to class them with depressive melancholia, from which they are difficult to distinguish clinically." Apart from senile, epileptic, post-apoplectic, alcoholic, and paralytic dementia, there are "certain definite clinical forms of psychoses which show a tendency to terminate in dementia, if they do not recover. In this respect all forms of motility-psychoses, especially the akinetic type, are not so very far from the paralytic psychoses. In the description of the special types (paralytic, post-apoplectic, epileptic, alcoholic, hebephrenic, senile) he says: "Hebephrenic dementia has the general marks of an immature state of development corresponding to the age of puberty. Hence the childish and silly behavior of these patients especially insisted upon by Kahlbaum and Hecker. Next to this, I should like to lay stress upon pronounced thoughtlessness or mental laziness as specific features of hebephrenic dementia. This is often shown in a very characteristic manner. The patients answer questions hit or miss without noticing their self-contradictions. The same question first answered with yes, may be later answered with no, or with a most general phrase. Gradually in the course of the examination an unmistakable annoyance at being forced to mental exertion is exhibited, and on questioning one may at times learn that thinking tires the patient. A turning away or the exclamation, "let me alone!" or even at times an outbreak of violence, puts an end to the interview. The attention of these patients is difficult to gain and especially difficult to hold for the purpose of examination. The form of observation is sometimes surprisingly good if judged by the patient's remembrance of casual incidents. One sometimes experiences surprises with these patients inasmuch as they casually betray fragments of knowledge, for instance, of languages or other acquired material of memory for which one searches in vain in a systematic examination. The facial expression, although at times more or less changed, usually remains much more vivid than might be expected from the degree of dementia (an instance in Heft 2, case 28, of the "Krankenvorstelungen"). The marks of mannerisms and oddity of expression are in most cases well defined, as is easily intelligible from the age of the patients; but according to my experience it is not as characteristic for hebephrenia as the intellectual laziness mentioned first."

A special "*secondary dementia*" is not described by him. But the admission of primary dementia in the sense of Kraepelin makes one ask: Why not recognize and extend the principle? These cases share the fate of the "paralytic psychosis." Among Wernicke's descriptions one looks in vain for the simple demented forms of general paralysis, whereas they form the background of the whole picture of the disease as furnished by Kraepelin.

After what we have learned of Ziehen's, Mendel's, and Wernicke's views, on "secondary dementia," let us start again from the disorder that is commonly so denominated by the average hospital physician. In the statistics published by the Lunacy Commission of the State of New York we find the following table:

Since October 1, 1888.

	ADMITTED.	RECOVERED.	DIED.
Mania, acute delirious	150	24	102
Mania, acute	9,816	4,109	1,293
Mania, recurrent	1,004	436	94
Mania, chronic	4,262	208	1,249
Melancholia, acute	15,400	5,110	2,013
Melancholia, simple	547	192	23
Melancholia, chronic	4,384	289	1,423
Alternating (circular) insanity	138	9	6
Paranoia	674	24	43
General paralysis	4,128	1	3,113
Dementia, primary	1,295	214	488
Dementia, terminal	13,170	107	7,733
Epilepsy with insanity	2,073	42	887
Imbecility with maniacal attacks	1,097	10	147
Idiocy	145	. .	33
Not insane [1]	473	. .	13
Unclassified	937	. .	. .
Total	59,693	10,775	18,660

This table shows that a relatively large number of cases figures among the acute psychoses which would be placed among the chronic or compound psychoses of the above-mentioned writers, if we assume that the recovery rate is about the same the world over, and not worse in New York. Of the acute psychoses as Ziehen recognizes them, at least 70 per cent. recover; of the combined *acute* manias and melancholias in the State of New York, only 36.5 per cent.

Leaving utterly out of the question the problem of disease-entities and treating the congregate mass of one hundred consecutive admissions to a hospital purely from the point of view of symptom-complexes, and of the final outcome, we may ask: In how many cases was it possible to predict with certainty the outcome? To what extent do the discrepancies point to insufficient inquiry into the facts? And to what extent is the satisfaction with vague terms at the bottom of the indifference to facts?

As a matter of fact, every alienist of experience will point to patients in whom secondary dementia can be foretold. Unfortunately, reasons are but rarely given. This is, however, one of the best methods of training in psychiatry: *To formulate in exact terms the facts which guide us in prognosis, to give the reasons why they do so, and to test these reasons on the basis of the final results.*

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[1] Includes cases of alcoholism, drug habit, etc.

In *general paralysis* we can say that there is a certain neurological status combined with a definite mental picture; that it points empirically to what may be called a diffuse myelo-encephalitis; that the type of distribution and the depth of the symptoms furnish evidence of a certain loss; and that empirically on such a basis a number of eventualities are to be expected.

In *delirium tremens* we have a certain standard combination of symptoms, mental and physical, and in drawing our conclusion we consider these facts in the light of our experience—the general make-up of the patient, and the possibility of a symptomatic delirium tremens (a phase of general paralysis, etc.).

In *acute excitement* we have our psychic and physical symptom-complexes and fragments of the etiological constellation to go by. In a fair number of the cases we will predict recovery; in others we remain in doubt; in yet others we predict an unfavorable outcome—death, or imperfect recovery, or danger of relapses or recurrences, or secondary paranoia, or secondary dementia.

The question now comes up—and it has been answered in the affirmative by Kraepelin: Are the signs which determine a good or a bad prognosis fundamental enough to build on them a grouping of mental diseases, which would be more natural than the present hazy plan or the more ambitious psychological schemes?

The clinical signs are of varying value. The nearer they are to the fundamental part which is diseased, the more direct is their bearing; the more accidental they are, the plainer their nature as mere circumstantial evidence, and the less convincing the claims of decisiveness. The symptoms are empirical and must stand the test of experience in order to command our attention. They must be judged in every case for what they are worth. Yet when we look at the average hospital record or at the average text-book description we fail to find the necessary helps for the distinctions which Kraepelin makes.

Wernicke's descriptions make a laudable exception, and in many respects they are more helpful methodically and less dogmatic than Kraepelin's composite pictures. We have seen that his mania, confused mania, and in part the hyperkinetic motility-psychosis form the excited phases of manic-depressive insanity, and affective melancholia and certain cases of depressive (akinetic) melancholia the depressive phases. The mixed phases are noticed by Kraepelin only. In hyperkinetic and akinetic states in which access to the inner life of the patient is difficult, and in the acute delusional and delirious states, a prognostic diagnosis appears much more problematic if based on the symptoms only. There is, however, so much harmony in what has been achieved by Kraepelin and Wernicke that we may look to the recent products of psychiatric work with the feeling that the confusion of words and the appearance of abstruseness will cease more and more in a measure as we keep to observation of actual cases, and records which cover the useful distinctions.

Viewing all these ways of getting at the facts of psychiatry, we cannot avoid the conviction that much of what floats in the air as solid tradition is, after all, very far from a firm foundation, and even far from doing justice to what we think and do in actual contact with cases. Much of it is due to an attempt to appear connected, orderly, and reasonable, in lecturing and writing, but it is apt to prove a real obstacle to progress in our work. If these traditional ideas are taken as suggestions, they are helpful; if taken as laws, they are temptations for a healthy law-breaking instinct, or they become harmful chains. In closing this review there are two warnings to which the reader's attention should be called. The first is: Beware of definitions which one is tempted to make from the sound of the words! Melancholia, dementia præcox, catatonia, represent totally different trains of thought in the various writers. The second warning is: Beware even of the definition given by any special author! It is better to study the histories of his cases, because his definition may unconsciously render the trend of words as he is accustomed to use them. It is not too much to say that it would often be difficult for these authors themselves to determine exactly where they stand in regard to some of the matters still in dispute. Their cases are more truthful, actual, and clear—when described in toto—than any attempt at a general psychopathology could be at the present time.

This review is necessarily merely a fragment of what occupies modern psychiatry, and fully intelligible in its bearing only to those who study psychiatry with the help of methods unfortunately too long neglected in our hospitals. Under the influence of pressure for financial results, and owing to the lack of centers for psychiatry instruction, very little material is collected in this country on which to bring the questions of the day to a test.

Psychiatry is the description and analysis of diseases which take months and years for their evolution, and which require careful records of conscientious and methodical observations in order to replace impressionist methods by an approximation to methods of precision. In this direction the above abstracts will certainly be suggestive. The review has, I hope, demonstrated that there are many questions about our patients which the average work on psychiatry does not attempt to answer, but for which good methods of analysis present helps. It has, I hope, convinced the reader that we need less discussions of generalities and more records of well-observed cases, and especially records of lifetimes, not merely snatches of picturesque symptoms or transcriptions of the meaning of traditional terms. It includes, as well as can be done in so little space, the chief standpoints and principles of work of the most productive pioneers of the day, and it will encourage many a reader to go to the originals, and say where to find things. It would seem that the time is not very far distant when the traditional headings of chapters in our text-books will change rather fundamentally, and to the great advantage of the student and practitioner.

CHAPTER II.

GENERAL ETIOLOGY OF INSANITY.

THE proportion of the insane to normal individuals may be stated to be about 1 to 300 of the population, though this proportion varies somewhat within narrow limits among different races and countries. It is probable that the intemperate use of alcohol and drugs, the spreading of syphilis, and the overstimulation in many directions of modern civilization have determined an increase difficult to estimate, but nevertheless palpable, of insanity in the present century as compared with past centuries.

The amount of such increase might easily seem to be large, on superficial examination, because of the imperfection of census-taking in the past, the accumulation of the chronic insane, and in new communities the constant upbuilding of new asylums.

Sex.—As regards sex, women and men are about equally affected, for the particular etiological factors determining insanity in the one (such as the puerperal period, the menopause, etc.) are evenly balanced by the special causes acting upon the other (struggle for existence, drunkenness, syphilis, etc.), and both sexes are about alike in their susceptibility to the two great etiological elements in alienation of the mind —heredity and mental or bodily strain.

Age.—The question of age is of great importance in a study of the etiology of insanity. While individuals are liable to mental aberration at any age, yet there are particular periods of life characterized by special vulnerability. In general, it may be said that this vulnerability is greatest in women between the ages of twenty-five and thirty-five, and in men between twenty and fifty, for it is at middle age that we find the maximum accumulation of etiological factors. But there are physiological epochs that influence markedly the line of psychic morbidity, and these are the periods of puberty and adolescence (fourteen to twenty years), that of genital involution in women (forty-five), and that of senile involution (sixty to seventy years).

But the chief factors in the causation of insanity may be summed up in two words—heredity and strain. The former is responsible for instability of the nervous system, the latter is multiform in character, comprising all of the stresses, physical and mental, direct and indirect, autochthonous and environmental, which may undermine the nervous constitution and bring it to its point of collapse.

Heredity.—In determining the factor of heredity we must not be content with ascertaining the existence of psychoses in the ascendants, but must seek, by careful interrogation of various members of the family, for some of the hereditary equivalents, such as epilepsy, chorea, hysteria, neurasthenia, somnambulism, migraine, organic diseases of the central nervous system, criminal tendencies, eccentricities of character, drunkenness, etc., for these equivalents are interchangeable from one generation to another, and are simply evidences of instability of the nervous system. It is the unstable nervous organization that is inherited, not a particular neurosis or psychosis, and it must be our aim in the investigation of the progenitors to discover the evidence of this.

That the statistics of insanity as regards heredity are often faultily gathered is too well known. In the first place, the recorder of the history of a patient frequently neglects to extend his inquiry far enough to include all of the transmissible psychoneuroses, and, in the second, the relatives are prone to conceal any supposed hereditary taint in the family. Here, for example, is a table prepared by the Lunacy Commissioners, showing the causes of insanity in 136,478 admissions to asylums in England and Wales, in which I find the item " hereditary influence ascertained " 20.5 per cent. Surely, so small a figure does not represent the true proportion of heredity as an etiological factor !

It will take many decads of much more careful compilation of histories to establish the actual ratio, but we shall attain nearer to the facts year by year.

No one has better formulated the principles of heredity in relation to insanity than Mercier,[1] who points out, among other things, that, besides the importance of the direct transmission of an unstable nervous system, there is another law of heredity, which is known as the law of sanguinity. Two parents may be perfectly stable and have normal organisms, and yet produce offspring with unstable and abnormal nervous constitutions, because of the unsuitability of the sexual elements of the parents to each other. The perfect organization of the progeny is the result of three factors—the quality of the germ (which brings matter), the quality of the sperm (which brings force), and the suitability of the one to the other.

The laws of heredity as they relate to insanity may be summarized briefly as follows :

1. The child tends to inherit every attribute of both parents.
2. Contradictory attributes can not be inherited from both parents.
3. The child may inherit the attributes of either parent solely.
4. It may inherit the qualities of one parent in some respects and of the other in other respects.

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[1] "Sanity and Insanity."

5. It may inherit the father's attributes for one period of existence and the mother's for another.

6. Some attributes have the quality of prepotency, or the tendency to push aside or overrule other attributes.

7. Attributes which are similar in both parents tend to become prepotent, giving rise to convergent or cumulative heredity.

8. Attributes may be transmitted in latent form from one generation to another, to reappear in a third or fourth or still more remote generation—a phenomenon termed "reversion."

9. Attributes tend to appear in the progeny about the same time of life at which they became manifest in the parents.

10. Attributes of the father tend to be inherited by the sons and of the mother by the daughters.

A study of the above laws will explain many of the puzzling features of psychopathic heredity,—why, for instance often only a few of the children of a neurotic parent suffer from neuroses or psychoses, and why psychoneuroses may develop in the progeny of healthy parents (latency). It must be remembered, too, that there is a variation in the degree of hereditary taint originated by the several heritable equivalents. Thus, simple neurasthenia, eccentricity of character, and a puerperal or senile psychosis are not so serious a heritage as epilepsy, chronic alcoholism, paranoia, and imbecility. The taint in a family is greater the larger the number of members and branches afflicted. When the degree of hereditary taint is marked, the psychoses which may develop tend to be modified from the ordinary types of such psychoses, and this deviation is termed hereditary degenerative modification,—or, in short, hereditary degeneracy,—while the insanity evolved is designated as a degenerative psychosis. The particular degenerative psychoses are such forms as idiocy, imbecility, feeble-mindedness, periodical and circular insanity, hysterical insanity, acute simple paranoia, polymorphic insanity, etc. A polymorphic course is particularly characteristic of psychic degeneracy, so that sometimes a perfect chain of psychopathic conditions and psychoses will be manifested throughout the life of the degenerate.

The polymorphism of hereditary transmission sometimes manifests itself in what is known as progressive hereditary degeneracy. For example, drunkenness in one generation may lead to simple psychoses in the next, to complex degenerative psychoses, epilepsy, etc., in the third generation, and finally, in the fourth, to idiocy, sterility, and the annihilation of the stock.

The indications of degeneracy in an individual are termed the stigmata of degeneration, or stigmata hereditatis. They may be defined as anatomical or functional deviations from the normal, which in themselves are usually of little importance as regards the existence of an organism, but are characteristic of a marked or latent neuropathic disposition. Much study has of late years been devoted to these indices by many investigators, particularly in their relation to insanity, idiocy, and criminal anthropology, and it behooves all who have to do with the development and care of the human body in any particular—and this refers especially to men of the medical and allied professions—to familiarize themselves with these signs of degeneration, in so far as they concern their own special provinces of work. These stigmata are vices of functional and organic evolution. The deviations from the normal may be in the way of excesses or arrest of development. They must be distinguished from the deficiencies or deformities produced by accidents at birth or by disease. I have said that these stigmata are anatomical and functional, but it is more convenient to divide the functional group into physiological and psychic classes. It is the latter which we are more apt to observe in our social relations with degenerate individuals. The psychic stigmata are always characterized by want of balance or lack of proportion between certain undeveloped or excessively developed faculties and other faculties which are normal. Defect of moral sense, of attention, of memory, will, judgment, or unbalanced excess of musical or mathematical aptitudes may be cited as instances of psychic stigmata. Hence the three following divisions may be made of all the degenerative indices: (1) Anatomical stigmata; (2) physiological stigmata; (3) psychic stigmata.

ANATOMICAL STIGMATA.

- Cranial anomalies.
- Facial asymmetry.
- Deformities of the palate.
- Dental anomalies.
- Anomalies of the tongue and lips.
- Anomalies of the nose.
- Anomalies of the eye:
  - Flecks on the iris; strabismus; chromatic asymmetry of the iris; narrow palpebral fissures.
  - Albinism.
  - Congenital cataracts.
  - Microphthalmos.
  - Pigmentary retinitis.
  - Muscular insufficiency.
- Anomalies of the ear.
- Anomalies of the limbs:
  - Polydactyly.
  - Syndactyly.
  - Ectrodactyly.
  - Symelus.
  - Ectromelus.
  - Phocomelus.
  - Excessive length of the arms.
- Anomalies of the body in general:
  - Herniæ.
  - Malformation of the breasts, thorax.
  - Dwarfishness.
  - Giantism.
  - Infantilism.
  - Feminism.
  - Masculinism.
  - Spina bifida.
- Anomalies of the genital organs.
- Anomalies of the skin.
  - Polysarcia.
  - Hypertrichosis.
  - Absence of hair.
  - Premature grayness.

PHYSIOLOGICAL STIGMATA.

- Anomalies of motor function:
    - Retardation of learning to walk.
    - Tics.
    - Tremors.
    - Epilepsy.
    - Nystagmus.
- Anomalies of sensory function:
    - Deaf-mutism.
    - Neuralgia.
    - Migraine.
    - Hyperesthesia.
    - Anesthesia.
    - Blindness.
    - Myopia.
    - Hypermetropia.
    - Astigmatism.
    - Daltonism.
    - Hemeralopia.
    - Concentric limitation of the visual field.
- Anomalies of speech:
    - Mutism.
    - Defective speech.
    - Stammering.
    - Stuttering.
- Anomalies of genito-urinary function:
    - Sexual irritability.
    - Impotence.
    - Sterility.
    - Urinary incontinence.
- Anomalies of instinct or appetite:
    - Uncontrollable appetite (food, liquor, drugs).
    - Merycism.
- Diminished resistance against external influences and diseases.
- Retardation of puberty.

PSYCHIC STIGMATA.

- Insanity.
- Idiocy.
- Imbecility.
- Feeble-mindedness.
- Pavor nocturnus.
- Precocity; one-sided talents; disequilibration.
- Eccentricity.
- Moral delinquency.
- Sexual perversion.

Having made this attempt to classify the various stigmata, we may now proceed to examine them in some detail:

Cranial Anomalies.—The most important features to be noted in connection with the head are asymmetry and a variety of deformities. It is necessary to an understanding of these stigmata to go over briefly a few facts of craniometry and cephalometry.

A score or more of distinguished anthropologists of the present century have been trying to discover racial distinction in human skulls; but the fact is that there are not so many characteristics of race in the cranium as in other parts of the body, and, accordingly, there are still wide differences of opinion as regards a scientific craniological classification. Races have been mingling so many thousands of years that cranial dissimilarities are the rule among them, even in tribes, and to some extent in families. These diversities of form have been designated as dolichocephalic, mesocephalic, and brachycephalic—words which merely convey an idea of the relation of the length to the breadth of the skull when viewed from above. The anteroposterior is to the biparietal diameter as 100 is to x, is the formula for determining this "cephalic index." All length-breadth indices below 78 are considered dolichocephalic; from 78 to 80, mesocephalic; and above 80, brachycephalic. We may assume that the physiological limits of this index are 70 to 90. This is based upon thousands of measurements of skulls by various investigators. Any excess or diminution of these figures must hence be regarded as pathological (Fig. 269).

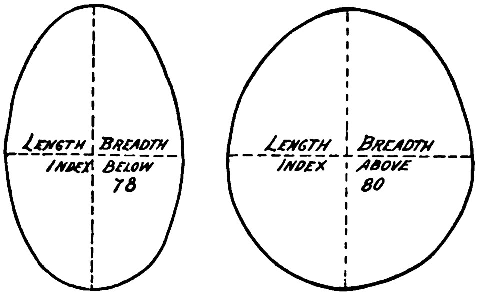


Fig. 269.

But while one skull may be narrower or broader than another, there is compensation in other diameters. The dolichocephalic has a greater vertical diameter, for instance, than the brachycephalic skull.

Besides these characteristics, something must be said regarding the physiological asymmetry of the skull. The fact that the arms and hands are not symmetrical on the two sides of the body, either in size or function; that the legs and feet are not symmetrical; that the left cerebral hemisphere is larger and more complicated than the right, would naturally lead us to anticipate some slight asymmetry of the two sides of the skull, and the facts of observation support us in the statement that asymmetry is the rule and perfect symmetry the exception. More than a thousand postmortem examinations, the examination of several hundred heads, and an inspection of some collections of skulls, such as that of Blumenbach, where I have particularly noted this point, together with the testimony of others, justify me in this assumption.

Asymmetry sometimes reaches extraordinary proportions,—often with quite a normal state of brain function, often with marked psychopathic changes. Outside of purely physiological asymmetry, we have that depending upon defective development and disease. One of the first of nature's constructive principles in fashioning the skull is the struggle of its contents for volume. Hence, as long ago pointed out by Virchow, premature synostosis of any cranial suture will lead to compensatory deformity. So, too, will arrest of developmen in any center of ossification, or a unilateral aplasia or hyperplasia of the skull bones, or of the contents of the skull.

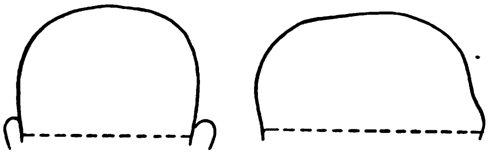


Fig. 270.—Chemocephalus.

Aside from the deformities of the head which are congenital in character, the diseases which most commonly produce cephalic deformation in early life are rachitis and hydrocephalus; in later life, tumors, exostoses, etc.; while at all periods of life the shape of the skull is menaced by injuries, from a forceps delivery to a falling brick. The following are some of the commoner designations of well-known cranial deformities:

Chemocephalus is flat-headedness. In this there is flatness at the top of the head. The condition is also called **platicephalus** (Fig. 270).

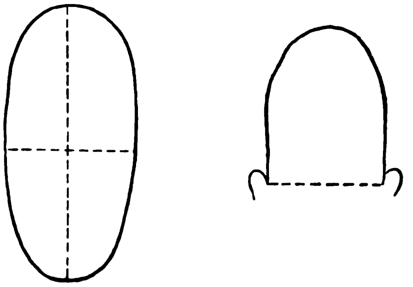


Fig. 271.—Leptocephalus.

Leptocephalus.—Early synostosis of the frontal and sphenoid produces leptocephalus, or narrow-headedness (Fig. 271).

Macrocephalus is a large head, usually due to hydrocephalus.

Microcephalus is a small head, due either to aplasia of the brain or premature synostosis of the sutures (rarely the latter).

Oxycephalus, or steeple-shaped skull, is due to synostosis of the parietal with the occipital and temporal bones, with compensatory development in the region of the bregma. Another name for this is *acrocephalus* (Fig. 272).

Plagiocephalus, or oblique deformity of the head, is due to unilateral synostosis of the frontal with one of the parietal bones (Fig. 273).

Scaphocephalus is probably caused either by too early union of the sagittal suture or by the development of both parietal bones from one center. The top of the head is keel-shaped (Fig. 274).

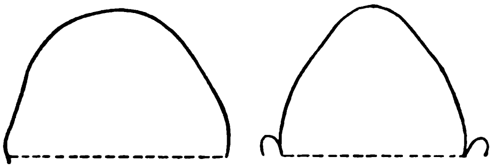


Fig. 272.—Oxycephalus.

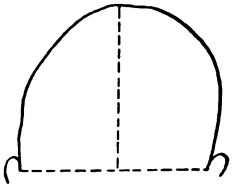


Fig. 273.—Plagiocephalus.

Trigonocephalus.—Premature union of the frontal suture, resulting in very narrow forehead and great width behind, giving rise to the term trigonocephalus (Fig. 275).

The two systems of measurement—the craniometrical and the cephalometrical—differ but slightly from each other, the former, of course, being the more exact, since every portion of the naked skull is attainable.

I would recommend the following series of measurements to be taken—eleven in number—in order to form a just idea of the capacity, shape, and symmetry of any head (Figs. 276 and 277): (1) The circumference; (2) the naso-occipital arc (*N* to *T*); (3) the nasobregmatic arc (*N* to β); (4) the bregmatolambdoid arc (β to *A*); (5) the binauricular arc; (6) the anteroposterior diameter (*S* to *O*); (7) the greatest transverse diameter (length-breadth index); (8) the binauricular diameter; (9) the two auriculobregmatic radii; (10) the facial length; (11) the empirical greatest height (B to β).

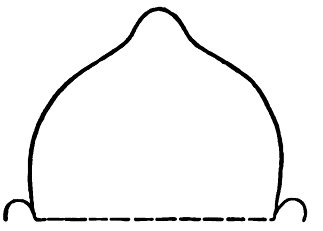


Fig. 274.—Scaphocephalus.

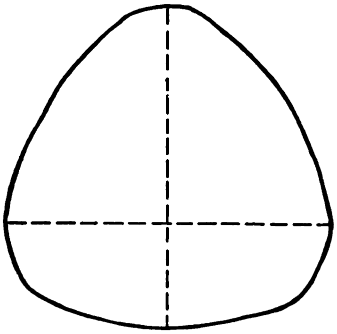


Fig. 275.—Trigonocephalus.

In addition to acquiring these mathematical data, cephaloscopic drawings are invaluable as exhibiting deformity clearly to the eye. Hence, the horizontal circumference, naso-occipital curve, and binauricular curve should be taken with a strip of lead, or, what is better, with the instrument devised by Luys (on the principle of the hatter's conformateurs), and the curves projected on paper.

Dolichocephalic heads, as a rule, have narrow, and brachycephalic have broad, faces. Something should here be said concerning prognathism, of which there are several forms. The best method of determining it is to measure the angle made by a line drawn from the nasal root to the junction of the inferior nasal spine and alveolar process (Fig. 277, *N* to *x*) with a vertical line dropped from the nasal root to Broca's horizontal. It is found that every normal skull exhibits this subnasal prognathism, but there is a wide variation in degree. Extraordinary prognathism, orthognathism, and opisthognathism—meaning extreme projection, straightness, or inclination backward of the subnasal line—are pathological.

The empirical greatest height of the head is an approximate measurement of the distance between the basion and vertex of the skull (*B* to β, or *U*). A line from the external occipital protuberance to the lowest median point of the superior maxilla, just above the incisors (*T* to *M*), passes almost directly through the basion. Hence, in cephalometry, by taking this diameter and the radii from each extremity to the bregma, we have a triangle (*M*, β, *T*) whose height (*B*, β) is easily ascertained. The height averages 13.3 cm. in men, 12.3 in women, and the physiological variation is from 11.5 to 15.

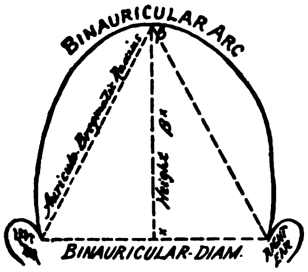


Fig. 276.

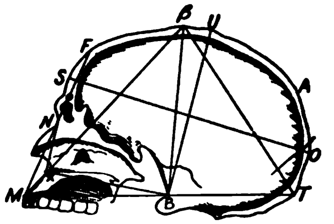


Fig. 277.

The only instruments necessary for obtaining the data just described are a pair of calipers, the tape-line, and a strip of sheet-lead two feet long by ½ or ¾ of an inch wide. Benedikt's calipers (manufactured by Wolters in Vienna), which are here illustrated, are to be recommended for their exactness (Fig. 278), as are also those that I have had made for my own use (Fig. 279).

Excessive prognathism is found among criminals, in microcephali, and in cases of hemi- and paraplegia spastica infantilis. Skulls known as crania progenæa have considerable pathological significance. In these, lower teeth project beyond the upper, and the inferior maxillary angle is obtuse, due, probably, to aplasia of the upper or hyperplasia of the lower maxilla.

The demonstration of the empirical greatest height is often quite valuable as an index of degenerative and neuropathic types. The following are some general points which should be considered in the examination of these cases:

A skull below the normal type in volume belongs to an abnormal individual.

Undertypical measurements of the head should always lead us to entertain the suspicion of defective cerebration.

Abnormal smallness of any part of the skull permits the conclusion that the part of the brain in its neighborhood is imperfectly developed.

Excessive development of the head has a double signification. It is always pathological, but may mean abnormality of brain or successful compensation. Wormian bones are also doubly significant. They either represent a pathological process or a successful effort of nature in repair.

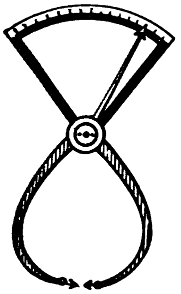


Fig. 278.—Benedikt's calipers.

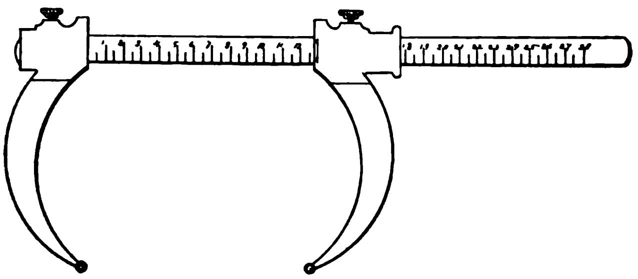


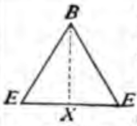
Fig. 279.—Author's calipers.

Hemiplegia spastica infantilis, epilepsy, and intellectual or ethic weakness often exhibit unilateral aplasia of the skull.

The skull is representative of the brain only during the years of its development, and it must be remembered that psychopathic deterioration often has its inception subsequent to the completion of the process, when no impression can be made upon its bony walls.

I have prepared a table of the measurements recommended, showing the averages in adults, male and female, together with the physiological variation, excesses above or below which are significant of morbidity. It is based upon the examination of some hundreds of skulls and heads, and upon statistics given by various authorities who have made especial study of this department of anthropometry. Hence it may be depended upon as a fair estimate of the dimensions of the head in most of the Caucasian races. The table is as follows:

TABLE OF CRANIOMETRICAL MEASUREMENTS.

	AVERAGE IN ADULT IN CENTIMETERS.		PHYSIOLOGICAL VARIATION.	REMARKS.
	Males.	Females.		
1. Circumference, ...	52	50	48.5–57.4	Roughly approximated, the volume is to the circumference as 1350 c.c. is to 50 cm.
2. Volume, ...	1500	1300	1201–1751	
3. Naso-occipital arc, ...	32	31	28–38	In figure, *N* to *T*.
4. Nasobregmatic arc, . .	12.5	12	10.9–14.9	*N* to β.
5. Bregmatolambdoid arc, .	12.5	11.9	9.1–14.4	β to *A*.
6. Binauricular arc, ...	32	31	28.4–35	
7. Anteroposterior diameter,	17.7	17.2	16.5–19	*S* to *O*.
8. Greatest transverse diameter	14.6	14	13–16.5	The formula for the length-breadth index is: Length : Breadth :: 100 : *x*. An index below 78 is dolichocephalic; 78 to 80, mesocephalic; above 80, brachycephalic.
9. Length-breadth index, . .	82.2	83.8	76.1–87	
10. Binauricular diameter, . .	12.4	11.9	10.9–13.9	 The height *B-X* of the triangle *E*, *B*, *E* formed by the auriculobregmatic radii and the binauricular diameter, averages 11.17 with a variation from 10 to 12.65.
11. Auriculobregmatic radii, .				
12. Facial length, ...	12.37	—	10.5–14.4	From root of nose, *N*, to lowest part of chin.
13. Empirical greatest height,	13.3	12.3	11.5–15	The empirical greatest height, *B*, β, is obtained by measuring the sides of the triangle *M*, β, *T*.

These measurements are those of the adult human skull. As the hair and scalp superadd about 3 cm., about 6 per cent. should be deducted in the head measurements Nos. 1, 3, and 6 to obtain those of the skull. In taking the diameters Nos. 7 and 8, deduct 1 cm. (the scalp averaging 5 mm. in thickness), and from the shorter radii, such as Nos. 10 and 11, subtract but 7 mm.

Facial Asymmetry.—Inequality of the two sides of the face—when congenital and not due to some such disease as hemiatrophy—is to be looked upon as a stigma of degeneration. In the same category may be grouped various irregularities, and such conditions as excessive prognathism or retrognathism. Great prominence or unequal prominence of the malar bones is to be observed, and also asymmetry of the orbits (Fig. 280).

Deformities of the Palate.—In connection with the soft palate, bifurcation of the uvula may be mentioned. As regards the hard palate, I have dwelt upon its deformities at some length in an article in the "International Dental Journal" (December, 1895), and the facts there brought forward may be recapitulated as follows:

While the palate occupies but a small place in this great category of hereditary stigmata of all kinds, it is one of the anatomical group, and this group is for many reasons the one of greatest importance. In this group, too, it occupies a distinctive place as being among the most striking, frequent, and significant of the anomalies.

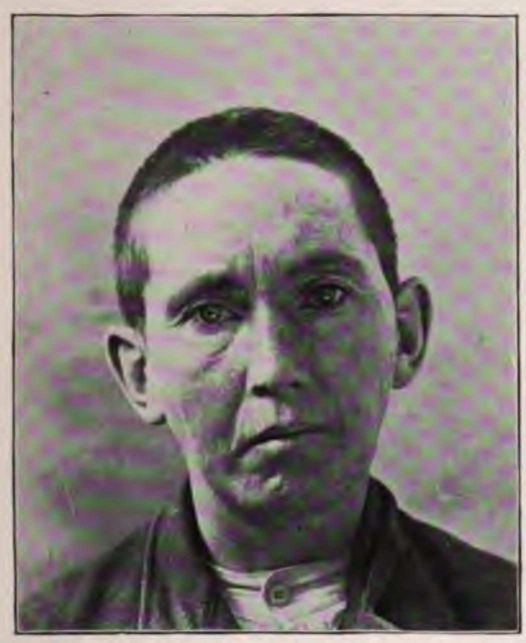


Fig. 280.—Male epileptic, aged forty years, with glabrous face and chin and facial asymmetry.

The arch of the hard palate presents considerable variation within strictly normal anatomical limits. A large, wide, moderately high vault is what may be called a normal standard. It means the highest evolution, judging from the fact that the mouth-cavity increases in capacity as we ascend the vertebrate series. Deviations from that standard are not at all infrequent, and yet such deviations may be normal. Thus, the palate may be low and broad, or it may be high and narrow; it may be short or long in its anteroposterior diameter; it may be ridged unduly along the palatine sutures, or it may present marked rugosities on its surface, especially in the anterior region; yet these variations are normal. Probably we may look upon these peculiarities as a species of compensatory development. Just as in a study of heads we find some very long and low, and others short and round and high, and recognize the fact that the shortness in one dimension is compensated for by a corresponding increase in another, so we may regard variation in palatine diameters.

The pathological palate has not been studied as much as it deserves to be. Save occasional and casual references to the "Gothic" palate in literature, and one or two papers upon the "torus palatinus," very little has been written upon the subject. In my paper, previously referred to, I have attempted to classify such pathological palates as could be justly looked upon as indicative of degeneracy. The word Gothic having been so long in use, and the hard palate being much like an arch or roof,[1] I have followed architectural nomenclature in the classification offered.

PATHOLOGICAL PALATES:

1. Palate with Gothic arch (Fig. 281).
2. Palate with horseshoe arch (Fig. 282).
3. The dome-shaped palate (Fig. 283).
4. The flat-roofed palate (Fig. 284).
5. The hip-roofed palate (Fig. 285).
6. The asymmetrical palate (Fig. 286).
7. The torus palatinus (Fig. 287).

The seven varieties named are to be looked upon as types merely. Each type will be found to present variations and combinations with other forms. Thus, the Gothic arch may have a low or high pitch and be short or long. The horseshoe arch (a familiar one in Moorish architecture) is always easily distinguished, but, owing to its conformation, a cast can not well be taken of it to show it in a perfect outline. The dome-shaped palate may be high or low, may be combined with asymmetry or torus. The presence of a torus in the Gothic variety is apt to destroy the purely Gothic form, and may cause it to resemble the flat-roofed palate. Under the heading of flat-roofed palate I should include all such palates as are nearly horizontal in outline, as well as those with inclined-roof sides but flattened gable. In the hip-roofed palate we have the sloping sides as usual, but also a marked pitch of the palate roof in front and behind; occasionally one may meet with a palate of this kind with so remarkable a pitch from before backward that it is almost like a Gothic roof turned about so that the gable runs transversely.

Asymmetry in the palate is commonly observed in many of the previously described forms, but occasionally is the only noteworthy peculiarity. It is usual to find asymmetry of the face and skull in cases with an asymmetrical palate. The torus palatinus (Latin *torus*, swelling) was first mentioned by Chassaignac as a mediopalatine exostosis. It is a projecting ridge or swelling along the palatine suture, sometimes in its whole length, sometimes in only a portion of its course. It is always congenital. It varies considerably in its shape and size, so that as many as five or six different species of torus are recognized. It may be wedge-shaped, narrow, broad, very prominent, or irregular. I have said nothing about cleft-palate, for I am not sure that it may be classed among the well-marked stigmata of degeneration. I have found but two or three cleft-palates among the 450 idiots and imbeciles on Randall's Island, while a number of cases of this kind with which I

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[1] "There is some confusion in literature of the roof of the mouth, or hard palate, referred to in this paper, with the dental arch, which is quite another thing."

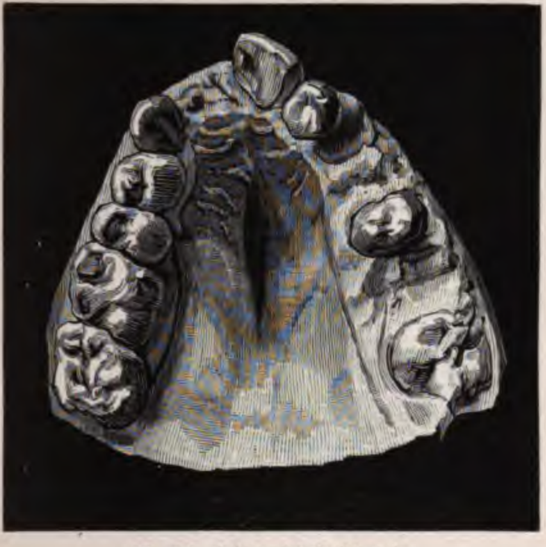

Fig. 281.—Palate with Gothic arch.

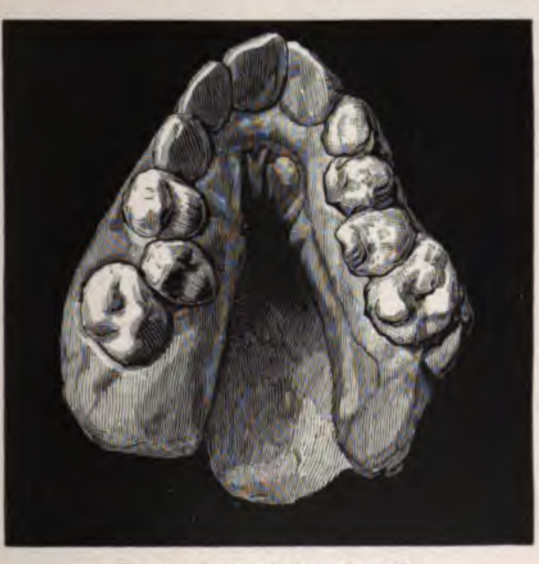

Fig. 282.—Palate with horseshoe arch.

have come in contact in my professional life were very far from degenerates. However, it would seem that there is great need of a faithful study of a large number of cases of cleft-palate in relation to the question of degeneracy. The deformed palate is, to my mind, one of the chief anatomical stigmata of degeneration.

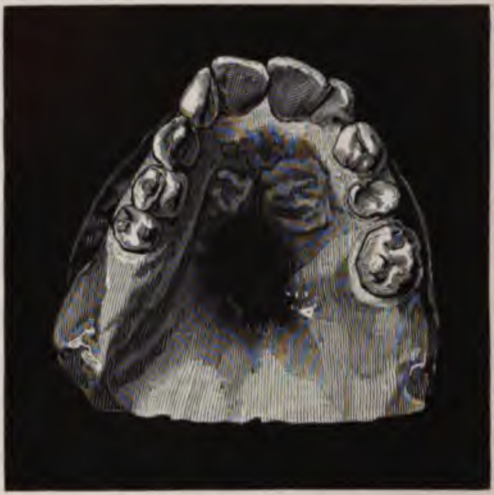

Fig. 283.—The dome-shaped palate.

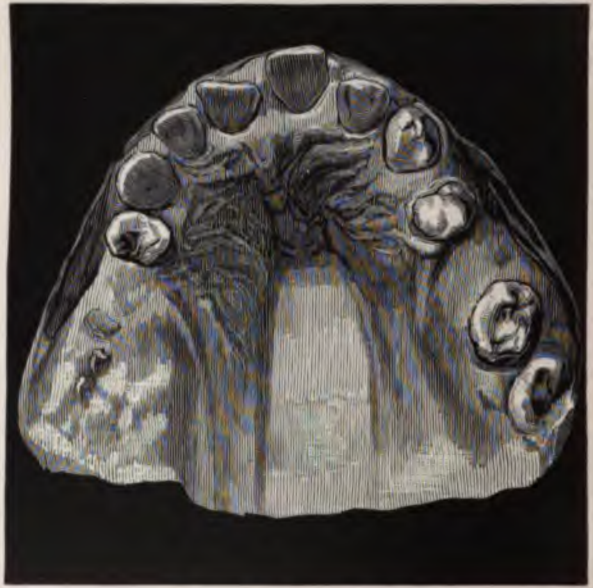

Fig. 284.—The flat-roofed palate.

It is true that, from this single indication, it would not be strictly scientific to adjudge an individual a degenerate. Occasionally, perhaps, a case presents itself where this anatomical stigma alone would suffice to insure a diagnosis of this nature; but usually other stigmata coexist, such as cranial anomalies, deformities of the ear, and the like. The frequency of the pathological palate among marked degenerates, such as the insane, idiots, and epileptics, has been testified to by many investigators. Thus, Talbot reported 43 per cent. of abnormal palates in 1605 inmates in institutions for the feeble-minded. Ireland makes it nearer 50 per cent. Charon, a later writer than these, found abnor-

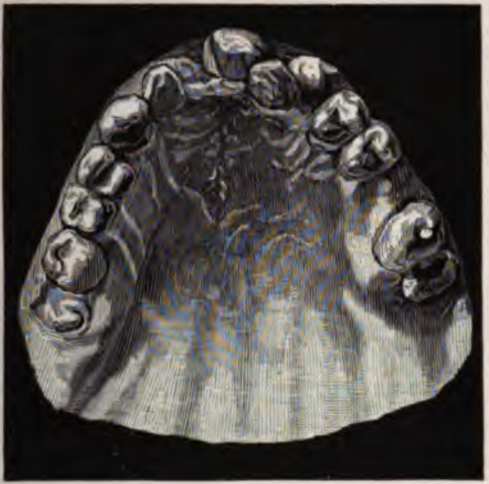

Fig. 285.—The hip-roofed palate.

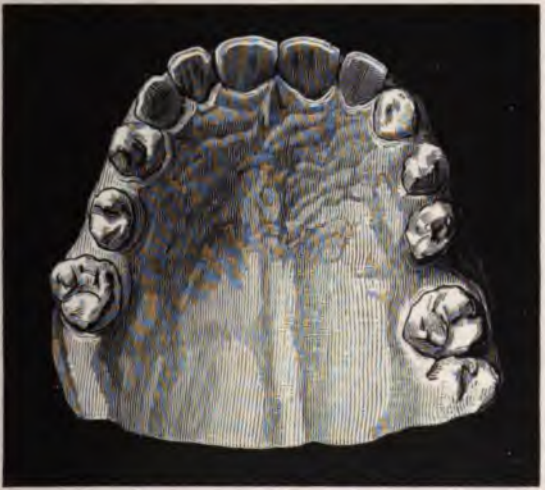

Fig. 286.—The asymmetrical palate.

mal palates in 10 per cent. of apparently normal persons, in 82 per cent. of idiots and feeble-minded, in 76 per cent. of epileptics, in 80 per cent. of cases of insanity in general, in 70 per cent. of the hysterical insane, and in 35 per cent. of cases of general paralysis. Näcke has studied particularly the torus palatinus in 1449 individuals, normal and psychopathic; he found it present in 23.9 per cent. of psychopathic women (insane, epileptic, idiot, and criminal), 32.9 per cent. of epileptic women, 34.4 per cent. of criminal women, 22.7 per cent. of normal women. The percentages were smaller in men than in women. A narrow torus is more common than a broad one.

Stieda examined 1500 skulls for the torus from an anthropological point of view. The skulls were of Prussians, Armenians, Africans, Frenchmen, Russians, and Asiatics. He decided that it has no anthropological significance; gives no racial distinction. While the torus is undoubtedly of value as an index of degeneration, particularly where it is well marked, it probably has less importance in this respect than some of the other forms of pathological palate.

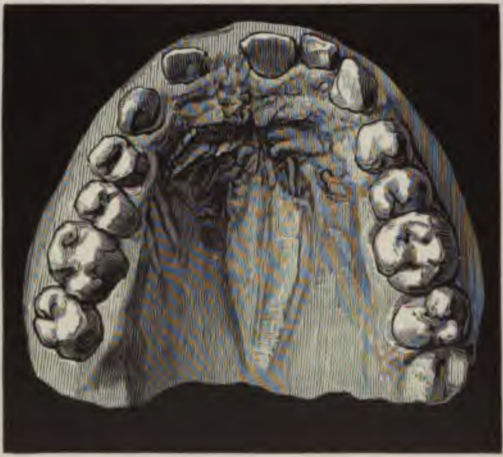

Fig. 287.—Torus palatinus (broad, wide torus).

Dental Anomalies.—Among anomalies of the teeth are macrodontism, microdontism, projecting teeth, badly placed or misplaced teeth, double row of teeth, or teeth which are striated transversely or longitudinally. Caries of the teeth and Hutchinson's teeth are due to neglect or disease. The latter, however, may often be considered as a stigma of degeneration. Then there is a retardation of the first and second dentition.

Anomalies of the Tongue and Lips.—A very large tongue (macroglossus) is not infrequently observed among the lowest classes of degenerates, as in idiocy. Sometimes there is microglossus, asymmetry of the two halves, or bifidity of the point. Harelip is somewhat more common than cleft-palate, but, like the latter, its exact standing as a degenerative stigma is not fully determined. Undue swelling or puffiness of the lips is noteworthy.

Anomalies of the Nose.—Marked deviation of the nose to one side or the other should be noted. Taken alone it may possess little significance, but in conjunction with other stigmata it is of value. The nose may be absent, or present defect of osseous development (*nasus aduncus*) or atresia of the nasal fossæ.

Anomalies of the Eye.—The pathological conditions of the eye have been placed in two groups in the foregoing classification, since some are anatomical and some physiological. To enumerate them altogether, they are as follows:

ANATOMICAL.	PHYSIOLOGICAL.
Flecks on the iris.	Blindness.
Strabismus.	Myopia.
Chromatic asymmetry of the iris.	Hypermetropia.
Narrow palpebral fissures.	Astigmatism.
Albinism.	Daltonism.
Congenital cataracts.	Hemeralopia.
Pigmentary retinitis.	Concentric limitation of the visual field.
Microphthalmos.	Nystagmus.
	Muscular insufficiency.

It is true that any one or two or more of these conditions present do not certainly indicate degeneracy, but they are significant in connection with other abnormal states, and all of them are more frequently observed in degenerate individuals, especially the lower orders, than in normal persons. In idiots, convergent strabismus, due to defect of refraction and in conjunction with hypermetropia, is very common. Muscular insufficiency and nystagmus (lateral or rotatory) are also often met with in this class of cases. In paralytic and other idiots and imbeciles homonymous hemianopsia is sometimes met with.

Anomalies of the Ear.—Deformities of the ear have been deservedly well studied, for as stigmata of degeneration they take high rank, like anomalies of the hard palate, in the anatomical group. Morel, Stahl, Wildermuth, Binder, and, more recently, Schwalbe, have given us especially good studies of these conditions. From their writings and my own studies, the following classification (following Binder) into twenty-two varieties may be made:

I. Abnormally implanted ears; they project too far or lie too closely, are placed too high or too low, too far forward or too far backward on the head.

II. Excessively large ears: (1) absolutely too large; (2) relatively too large in small or microcephalic individuals.

III. Ears which are too small.

IV. Too marked conchoidal shape of the ear. The details of the ear (anthelix and crura, etc.) are but slightly marked, while the helix outlines the ear like the rim of a funnel.

V. Ears which have a general ugly shape. The breadth of the upper part may exceed that of the lower, and vice versâ; excessive length; ears without lobules; unusually short ears.

VI. Ear not uniform in width; usually a long ear with one or more constrictions in its breadth.

VII. The Blainville ear; asymmetry of various kinds of the two ears. In most cases the asymmetry is due to an anomaly of the left ear.

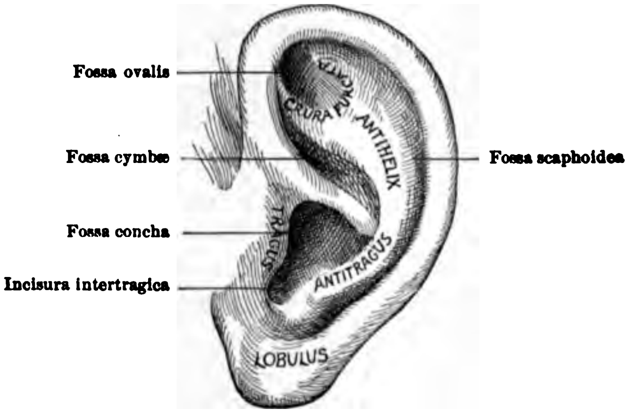

Fig. 288.—Normal ear.

VIII. The ear without lobule; there are usually other deformities of this ear besides the absence of lobule, such as too large a concha, prominence of the anthelix, etc.

IX. The ear with adherent lobule; the lobule is enlarged, adherent, and inclines downward toward the cheek.

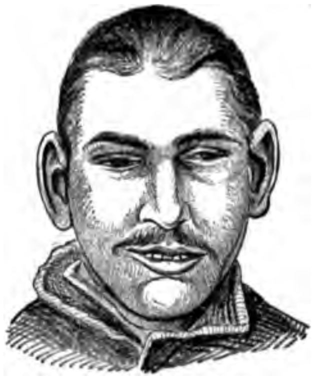

Fig. 289.—Blainville ear; also excessive length of ears.

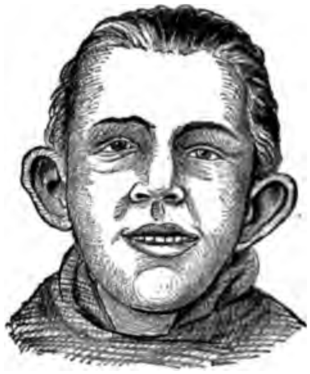

Fig. 290.—Morel ears.

X. The Stahl ear, No. 1.[1] A series of anomalies of the helix. The helix is broad, like a band, and coalesces with the cartilage of the crura furcata. The fossa ovalis and fossa scaphoidea are scarcely to be seen. The lower half of the helix is obliterated. There are occasionally slight variations from this type.

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[1] See "Zeitschrift für Psych.," vol. xvi.

XI. The Darwin ear; helix interrupted where its transverse portion passes into the descending, and at this point is a projection of the rim above and outward, like the pointed ear of lower animals.

XII. The Wildermuth ear.[1] The anthelix projects so far as to form the most prominent part of the auricle.

XIII. The ear without anthelix or crura furcata.

XIV. The Stahl ear, No. 2. Multiplication of the divisions of the crura furcata, so that there are three instead of two crura.

XV. Wildermuth's Aztec ear. Lobule wanting; the whole ear seems pushed forward and downward; the crus superius of the anthelix coalesces with the helix, while its crus anterius is scarcely perceptible.

XVI. The Stahl ear, No. 3. Only the crus anterius of the crura furcata is present, while the auricle seems divided into two halves by a ridge from the antitragus.

XVII. The ear with double helix.

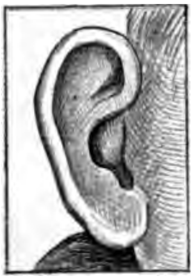

Fig. 291.—Stahl ear, No. 1.

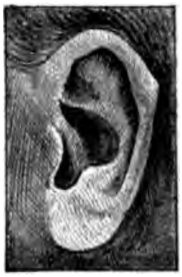

Fig. 292.—Darwin ear.

XVIII. The ear with too large or too small a concha.

XIX. The ear with continuous fossa scaphoidea. The fossa passes down into the lobe.

XX. The Morel ear. A form marked by abnormal development of the helix, anthelix, fossa scaphoidea, and crura furcata, so that the folds of the ear seem obliterated, and the ear is smooth, larger than usual, often prominent, and with thin edge.

XXI. Ears misshapen by abnormal cartilage development. Here belong all irregular cartilaginous growths and thickenings except those caused by hematoma of the ear.

XXII. Various peculiarities, difficult to classify, are included here, such as abnormalities of the semilunar incisure of the tragus and of the meatus, coloboma of the lobule, hairiness of the different parts of the auricle, accessory ears, clefts, etc.

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[1] "Wurt. Corresp.-Blatt," 1886, No. 40.

The most important malformations of the ear—those that may be regarded as belonging to the stigmata of degeneration, and those, too, which are striking and plain to the eye—are to be summarized as follows:

The deep position of the crus anterius.
Marked prominence of the anthelix.
Excessive broadening of the ear.
Stunted development of or absence of the helix.
Trifurcation of the anthelix.
Widening of the fossa scaphoidea.
Absence of the crus superius.
Complete absence of lobule.
Asymmetry of the two ears.
Excessive enlargement or diminution of the concha.
Excessive conchoidal structure of the ear.

Reference is occasionally made in literature to the Cagot ear. The Cagot is a species of cretin in the French and Spanish Pyrenees, in which one of the chief physical deformities is absence of the lobule of the ear.

Binder states that the adherent lobule exists in almost one-third of normal persons, and in the photographs of several hundred distinguished persons 15 per cent. had abnormal lobules. At the same time more than twice as many adherent lobules are found in degenerates as in normal individuals.

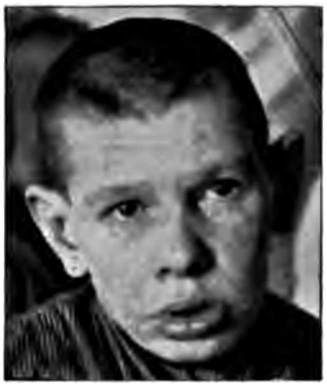
Fig. 293.—Excessive length of ears; facial asymmetry.

Now, with regard to statistics of malformed ears in degenerate individuals, Wildermuth noted this condition in 41 per cent. of 142 idiots. Binder found 64 per cent. of degenerate ears in 354 insane persons. It is to be remarked, however, that Binder was more careful in his examinations, and by long practice had acquired more expert knowledge than Wildermuth. Fränkel observed degenerate ears in 29 cases out of 32 with cranium progannæum.

Knecht found 20 per cent. of degenerate ears among 1274 criminals, 27 per cent. among 48 epileptics, and 32 per cent. among 84 insane.

Binder noted degenerate ears in 33 persons outside of institutions, supposed to be normal individuals. Inquiring closely into their histories, he discovered that 7 of them had insane parents, brethren, or children; in 19 there were decided psychic abnormalities, and only 7 were apparently normal persons. As regards heredity, it is very common for children to inherit ears with the identical characteristics of those of one or the other parent, but, on the other hand, it is not uncommon for the ears of the children to be quite different.

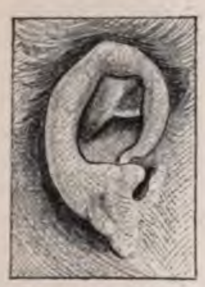

Fig. 294.—Broad, band-like helix; no anthelix; no lobule; excessive size of fossa cymbæ.

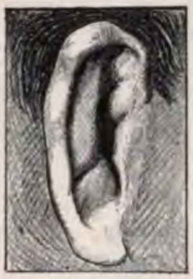

Fig. 295.—Excessive length of ear; fusion and distortion of helix, anthelix, antitragus, and lobule.

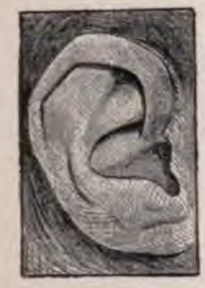

Fig. 296.—Triplication of crura furcata; malformed helix and antitragus; absent lobule.

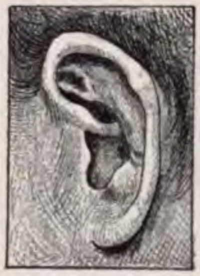

Fig. 297.—Fissure in anthelix; slight Darwin tubercle; slight antitragus.

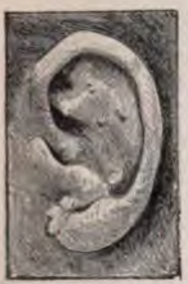

Fig. 298.—No crus superius; no anthelix; small fossa conchæ; few details of ear.

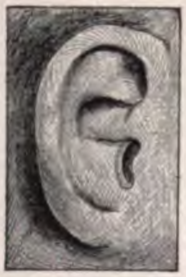

Fig. 299.—No lobule; no fossa conchæ; shallow fossa scaphoidea; fusion of helix, anthelix, and antitragus; a type of Stahl ear, No. 3.

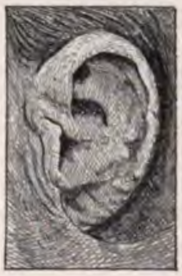

Fig. 300.—Prominent anthelix; maldeveloped helix; absence of lobule; diminution of the concha; Wildermuth ear, No. 1.

Anomalies of the Limbs.—Paralysis, atrophy, retarded growth, club-foot, and athetosis are conditions due to disease of the brain, and are observed in many cases of paralytic idiocy. These are not properly stigmata of degeneration, although they may be such under some circumstances, as, for instance, when club-foot or club-hand has a teratological origin. On the other hand, there are anomalies having a hereditary character, which are essentially degenerative indices. Among these may be mentioned congenital luxations, supernumerary fingers or toes (polydactyly), fusion of fingers or toes (syndactyly or aschistodactyly), excessive length of the arms as compared with the rest of the body and the lower limbs, missing fingers or toes (ectrodactyly), missing limb (ectromelus), fusion of the extremities (symelus or symmelus), or absence of parts of limbs so that they are excessively short (phocomelus). There may also be anomalous brevity of some digits as compared with the relative proportions of normal digits. Excessive volume of limbs (megalomelus) or digits (megalodactyly) or excessive gracility of limbs (oligomelus) or of digits (oligodactyly) also deserve mention.

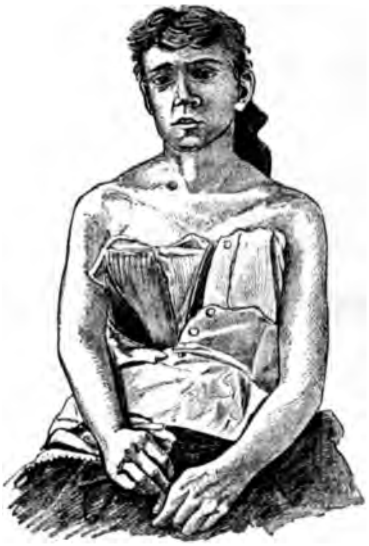
Fig. 301.—Phocomelus right arm in epileptic girl; right humerus several inches shorter than left; arms otherwise perfect.

Anomalies of the Body in General.—Local malformations are naturally of more importance than general anomalies of the whole form, but it is necessary to study the relative proportions of the entire figure from an anthropometrical point of view, and to compare the results with normal standards. Excessive diminutiveness of figure, as well as excessive or giant growth, are indications of degeneracy. So, too, are infantile characteristics in an adult, feminine peculiarities in males, and masculine traits in females. In this regard, observations of the relative proportions of the shoulders and pelvis are particularly useful. The occult form of spina bifida with local hypertrichosis is met with. Deviation of the vertebral column among neuropaths is mentioned by Féré. They may be lordoses, scolioses, or kyphoses in various degrees. The coccyx may present peculiarities, such as simulation of a tail. Thoracic asymmetry or other deformity is observed at times. Absence of pectoral muscles, or of muscles in various parts of the body, has significance. Herniæ are evidence sometimes of arrest of development of some part of the abdominal wall. Excessive development of mammary glands in males, or their absence or reduplication (polymastia) in either sex, constitutes an evidence of degeneracy.

Anomalies of the Genital Organs.—Among the genital anomalies in males are cryptorchismus; unilateral or bilateral microrchidia; spurious hermaphroditism; insufficient development of the entire genital apparatus; hypospadias; epispadias; defect, torsion, or great volume of the prepuce; median fissure of the scrotum; imperforate meatus.

In females the labia may be abnormally large, simulating a scrotum; sometimes very small. The clitoris may be exceedingly large. The labia minora may be hypertrophied. Sometimes there are intermediate folds between the labia minora and labia majora. The labia minora may be pigmented, particularly in brunets and when they are hypertrophic. There may be imperforate vulva, or atresia of the vagina, or double vagina; uterus bicornis is sometimes met with.

Anomalies of the genito-urinary apparatus should always be sought for, for, though most frequent among idiots, imbeciles, epileptics, and the like, they are by no means rare in other classes of degenerates and in degenerate families. In males, defect of the testicles often coincides with general excess of growth in the whole body or in the lower extremities, such as is often produced by castration in man and lower animals.

Anomalies of the Skin.—Among the anomalies of the skin are to be mentioned adipose thickening; polysarcia; precocious and often abnormal development of the hairy system; hair along the spinal column; rudimentary tail; premature grayness; a glabrous chin in grown men; persistent lanuginous character of the hair; excessive growth of hair on the chin and breast in women; complete or partial discoloration of the hair (albinism, vitiligo); local or general hypertrichosis; partial or complete absence or fetal state of the nails; melanism of the skin; pigmentary or vascular nevi; molluscum; ichthyosis; vitiligo; albinism; pigmented spots.

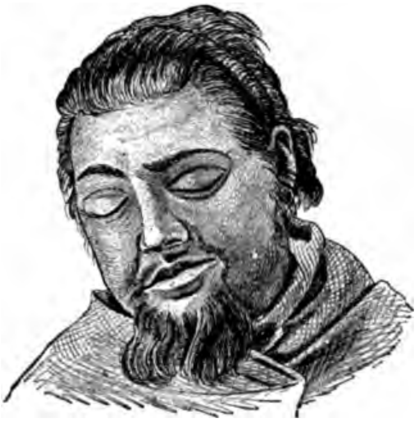
Fig. 302.—Hypertrichosis in a female imbecile.

Anomalies of Motor Function.—Delay in acquiring a knowledge of the proper use of muscles for walking, eating, and the like may often be regarded as an index of degeneracy. Where ordinary etiological factors may be excluded, tremors, tics, epilepsy, and nystagmus may have a similar value. Even when not congenital, they often indicate hereditary instability of the nervous system.

Anomalies of Sensory Function.—The numerous anomalies of function in connection with the eye have already been mentioned. Congenital deafness has also its significance. So, too, have hereditary forms of migraine and neuralgia. Certain defects or excesses in general cutaneous sensibility have been noted as frequent among degenerates. Thus several excellent writers on this subject have stated that a general anesthesia is not uncommon, especially among lower classes of degenerates. In some instances there is hyperesthesia.

Anomalies of Speech.—It may be questionable as to how far stammering and stuttering are to be looked upon as functional degenerative stigmata, but they are certainly found more often in children with a neuropathic inheritance than in children with good heredity. Delay in the acquisition of language and complete or partial defect of speech have more significance.

Anomalies of Genito-urinary Function.—Sexual irritability, impotence, sterility, and urinary incontinence must be considered as indices of neuropathic disposition. Retardation of puberty in both sexes, but especially in the male sex, is a noteworthy indication.

Anomalies of Instinct or Appetite.—It has been pointed out that, among all degenerates, there is a taste or appetite for certain foods or drugs which tends to favor their dissolution (alcohol, morphin, cocain, and the like). In many cases of inebriety the uncontrollable appetite is to be looked upon as a functional stigma of neuropathic inheritance. Gluttony, merycism, and the like are usually similar indications.

Miscellaneous.—A diminished resistance against external influences (such as strains of various kind) and diseases is significant. Great precocity of intellectual development and of certain aptitudes, and morbid emotional conditions, are among suspicious indications of a neuropathic basis.

The psychic stigmata of degeneracy need only the foregoing enumeration.

PHYSICAL AND MENTAL STRAIN.

At the beginning of this chapter I spoke of the etiology of insanity as being describable in two terms, heredity and strain—heredity, which renders the nervous organization unstable, the strain, which causes the unstable nervous centers to collapse. Doubtless there are limits of endurance in any organization, no matter how strongly balanced, if the strain be great enough, but the instances of insanity developing in individuals with properly balanced and adjusted nervous organizations are rare indeed. The strain which breaks the unstable nervous system is physical or moral, often both. What organism could withstand the assaults upon its integrity of all three of these factors—heredity, physical ill-health, and cankering care? It is difficult to estimate accurately the proportion of one cause as compared with another, since usually several are associated in the same case; but I believe that statistics will support me in the statement that the physical causes (in which I include alcohol, bodily diseases and disorders, accident and injury, old age, the puerperal state, the menopause, and the like) surpass the moral causes (grief, domestic trouble, business worry, overwork, religious excitement, love affairs, fright, nervous shock, etc.) as factors in insanity by about two to one—that is, twice as many are made insane by physical strain as by mental strain. It now behooves us to examine these divers stresses, and to show how some of them give a special color or character to the psychosis developed. It is best to present them somewhat in the order of their frequency, under two or three categories, the most common and most important first, the rarest last. The physical, physiological, and moral causes, then, will be considered in the following order:

Physical:
1. Toxic (autotoxins, alcohol, narcotics, metallic poisons, etc.).
2. Bodily diseases and disorders (syphilis, acute and chronic diseases of the nervous system).
3. Trauma to the head.
4. Nervous exhaustion.

Physiological:
1. Puberty.
2. Puerperal state.
3. Menopause.
4. Senility.

Moral:
1. Emotional strain.
2. Imitation.

Toxic Influences.—It is not surprising that deleterious agents in the blood, which bathes every cell and fiber of the nervous system, carrying thither the necessary nutritional elements and removing thence the waste products, should readily overstimulate, retard, pervert, or destroy its high functions. Some of these agents (like alcohol) also affect the nutrition of the central nervous system, by inducing disease of the arteries and of the stomach, liver, and kidneys. Some of the poisons cause insanity by long-continued chronic action upon the nervous system, and others by acute intoxication.

Auto-intoxication.—Accumulation of deleterious agents in the blood in such quantity as to affect the nervous system—*e. g.*, carbonic acid and the poison of diabetes and of uremia—has been long known to medical science; but the more mysterious poisons produced by disease in various parts of the body, by fermenting or putrefying substances in the alimentary tract, and by some of the acute infectious fevers, have only of late taken an important place in the etiology of the psychoses. We do not yet know how frequently auto-intoxication from absorption of intestinal poisons determines insanity, but the facts thus far collected point to the origin of a considerable number of cases from this cause. These cases are usually of depressed type, but sometimes maniacal.

Alcohol.—While the position of autotoxemia as a factor in etiology is not yet determined, we may say of alcohol that it stands foremost (after heredity) as a single, independent cause (eighteen to twenty per cent. in males). Acute alcoholism rarely induces a psychosis. Alcoholic insanity commonly develops from chronic alcoholism, from the excessive use of the poison for a long period of time. It is three or four times as frequent as a factor in the insanity of males as of females. Usually it is not difficult to discover the cause of an alcoholic insanity, but, should alcoholic abuse be denied, an investigation of the condition of the viscera will often throw light upon the subject (cirrhosis of the liver, fatty heart, chronic gastric catarrh with heavily furred tongue, chronic nephritis, and arteriosclerosis). Corroborative evidence will generally be afforded, too, by affections of the nervous system (alcoholic polyneuritis; alcoholic epilepsy; muscular paresis here and there in the hands, face, or tongue; fibrillary tremor of the face and tongue, fine or coarse tremor of the fingers and hands; paresthesias, hyperesthesias, neuralgias; muscæ volitantes, tinnitus aurium, amblyopia, and visual hallucinations). The peculiar psychic degeneration of alcoholism is very characteristic. This consists of gradually weakening memory and will, slowness of perception and judgment, and loss of esthetic and moral sense, with occasional states of depression and accesses of anger. The psychoses which develop upon this basis are marked by two or three features, which are considered rather pathognomonic:

1. Often a peculiar loss of the sense of time and place (a paramnesia).
2. A tendency to illusions and hallucinations, innumerable, changing, mobile, and variegated.
3. A tinge of weak-mindedness in the psychic symptoms presented.

Hysterical manifestations are not uncommon in alcoholic insanity.

Morphin.—Morphin is, among the alkaloids, the most frequent cause of insanity. It is a sad commentary on the heedlessness of some medical men, but the family physician is responsible in almost every case of development of the morphin habit and its far-reaching consequences. It should be looked upon as a sin to give a dose of morphin for insomnia or for any pain (such as neuralgia, dysmenorrhea, rheumatism) which is other than extremely severe and transient. The earliest symptom of morphinism is a general sensation of disquiet, manifested by incoherence of thought, difficulty of concentration of the mind, marked motor restlessness, and insomnia. The dose is gradually increased, and may reach a maximum of five or more grams.

The chief physical disorders induced by long-continued use of morphin or opium are as follows:

1. Anorexia and constipation (later, diarrhea often).
2. Cachectic anemia.
3. Cardiac weakness and intermittence, and bradycardia.
4. Muscular weakness with tremor.
5. Miosis in the early stages, mydriasis later, with sluggish reaction of the pupils.
6. Impotence. Amenorrhea in women.
7. The knee-jerks are often absent.
8. Diminished sensibility to touch and pain, and concentric limitation of the visual fields.
9 Headaches and localized shooting pains, neuralgias, and paresthesias.
10. Sensation of feeling cold.

The psychic symptoms may be summarized briefly, thus:

1. Simple elementary illusions and hallucinations, muscæ volitantes, tinnitus aurium.

2. Loss of will and esthetic sense, irritability; moral perversion, as in alcoholic psychic degeneration, but with little failure of memory.

3. Diminished attention, incoherence of ideas, and easily fatigued intellectual powers.

A well-developed psychosis is usually the result of abstinence from morphin, and not of the chronic misuse of it. It varies in degree up to a type approaching acute mania.

Cocain.—Of recent years there have been numerous instances of cocain insanity, and they are doubtless growing more frequent. While with morphin it is the abstinence that is prone to induce a psychosis, with cocain, on the contrary, it is the prolonged use of the drug that develops the insanity, while abstinence gives rise to few noteworthy symptoms. The misuse of cocain leads to the evolution of an acute hallucinatory paranoia.

Hashish (Cannabis Indica).—We never see insanity from this cause in America, but in Egypt and India it is extremely common. In visits paid by the writer to the Cairo Insane Asylum in the winter of 1891–'92, he observed 64 cases of the 248 patients in the institution in which the insanity was due to the inhalation of hashish by smoking. The symptoms produced are indigestion, diarrhea, increased appetite, dilatation of the pupils, drooping eyelids, anemia, general debility, and delirium. The earliest mental symptom is marked and increasing timidity, sometimes amounting to *folie du doute*, or an agoraphobia.

Atropin; Hyoscyamin; Hyoscin.—These isomeric alkaloids have much the same physiological effects (mydriasis, paralysis of accommodation, dryness of the throat, depressed heart's action, dreadful illusions and hallucinations, etc.), but instances are not common of their giving rise to psychoses. However, it is probable that the employment of one of these as a secret cure for drunkenness has been the cause of serious insanity in a considerable number of cases that have found their way from sanatoriums to asylums.[1]

Metallic Poisons.—Lead and mercury at times induce insanity, the former much more frequently than the latter. The intoxication is chronic, but the psychosis developed may be either acute or chronic. Both of these poisons produce similar psychic symptoms, such as vertigo, sleeplessness, rudimentary or marked hallucinations, confusion and incoherence, anxious depression, and often persecutory delusions. In severe cases there is dementia. In lead cases there are usually to be observed the concomitant physical symptoms, such as anemia, colic, blue line on the gums, tremor, arthralgia, palsies, and convulsions. In mercury cases we note stomatitis, tremor, and gastro-intestinal catarrh. Hysterical symptoms are not infrequently superadded upon the lead and mercury psychoses.

Various Poisons.—There are many other poisons which, in rare instances, produce insanity. Among these may be mentioned coal-gas, carbonic oxid, stramonium, henbane, hemlock, bisulphid of carbon, etc. The writer described some years ago three cases of bisulphid of carbon insanity which ran their course under the type of acute mania going on to recovery, studied by him at the Hudson River State Hospital for the Insane.[1] All three were workers in a rubber factory.

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[1] Dr. B. D. Evans, Superintendent of the Morris Plains Asylum, N. J., has collected a number of such cases.

Bodily Diseases and Disorders.—Syphilis.—Syphilis is one of the most important of the physical causes of insanity. It acts upon the brain indirectly through wide-spread, severe disturbance of general nutrition and through arteriosclerosis, and directly by the production of diffuse changes in the tissues of the central nervous system, or of circumscribed meningeal deposits or intracerebral gummata. The degeneration of cells and fibers, the gliosis and the arteriosclerosis, are possibly due to toxins created by specific micro-organisms, and not to the direct influence of the germs themselves, which may explain why syphilitic psychoses are ordinarily late manifestations of syphilis. General paralysis and cerebral syphilis are the chief phases in which the psychosis is presented. It is often difficult to obtain a history of syphilis in a patient, so that the statistics as to the frequency of syphilis as a cause of general paresis, for instance, are generally faulty. Where the history is uncertain, a careful examination may indicate the existence of syphilis (hereditary syphilis in the children, leukoderma, cicatrices, swelling of the lymph-glands, periosteal deposits and tophi, perforation of the palate, nasal symptoms, etc.).

Hereditary syphilis plays a part in the etiology of the psychoses of early life,—for example, imbecility and idiocy,—though probably not so great a part as is frequently asserted, for, in a considerable experience with such conditions at the Randall's Island Asylum for Idiots, I have seen but little hereditary syphilis.

Acute Infectious Diseases.—Typhoid fever, malaria, pneumonia, influenza, and acute articular rheumatism head the list of acute fevers which sometimes superinduce insanity. Disturbances of nutrition, high fever, and toxic changes in the blood are responsible for the symptoms developed. Perhaps the toxin-producing bacteria are the chief agents, acting by direct influence upon the cortical cells and fibers. If this be true, these cases were better classed under the head of Toxic Influences. At the height of a fever we have a febrile delirium, characterized by hallucinatory incoherence; but, later on, when the fever has diminished and the organism is weakened by disease, such manifestation is termed "inanition delirium." From either the febrile or inanition delirium a psychosis may develop, usually assuming the type of a hallucinatory paranoia with self-depreciatory or persecutory delusions, in some cases with a tendency to agitation, in others with inclination to a stuporous condition. Mania and melancholia are rare, stupidity with a proclivity to terminal dementia more common. Malarial psychoses sometimes exhibit a certain periodicity corresponding to the intermittent nature of the cause. Heredity, alcoholic degeneration, etc., also play a considerable part in the etiology of this form of toxic mental disorder.

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[1] "Boston Med. and Surg. Jour.," Oct., 1892.

Tuberculosis.—The disturbances of nutrition in tuberculosis, as well as the mental depression sometimes associated with the disease, occasionally lead to the development, out of an exhaustion or inanition delirium, of a true psychosis—a melancholia or a hallucinatory excitement. It may be said, however, that the relation of tuberculosis to insanity is much more frequently that of sequel than of prodrome, for many cases of melancholia and stuporous forms of insanity die of this disease owing to shallow respiratory functions and insufficient nutrition.

Carcinoma.—The progressive cachexia induced by malignant disease, as well as the direct effects of cerebral metastases, sometimes lead to psychopathic conditions resembling those of tuberculosis.

Heart Disease and Atheromatous Arteries.—Cardiac disease is frequently found among the insane, but its precise relation to the psychoses is obscure. Doubtless, in so far as it disturbs the circulation and interferes with cerebral nutrition, it predisposes to mental instability. On the other hand, disease of the arteries (senile, nephritic, syphilitic, alcoholic, cachectic), is a much more effective disturber of nutrition, and at the same time gives rise to serious focal lesions, such as miliary aneurysms, thrombosis, and hemorrhage, which may be etiologically associated with various psychopathies.

Nephritis.—The nephritic psychoses assume usually the type of a hallucinatory paranoia, and therein resemble other toxic insanities. It is probable that toxic changes in the blood are here of more importance than the changes in the vascular walls, though these, too, have their significance.

Gastro-intestinal Disorders.—These sometimes induce hypochondriacal melancholia, and predispose to psychoses of various kinds by disturbing nutrition; but their frequent relation to insanity is generally a consequence rather than a cause.

Diseases of the Genital Organs.—There are serious disorders of the female genital organs which occasionally play a rôle in the causation of insanity, but their importance as factors has been grossly exaggerated, and much harm and little good have followed operative interference for the relief of the insanity. Probably the cessation of menstruation (usual in acute insanities) has been misinterpreted as significant of genital disease, and thus given rise to a grave error.

I would not be understood as decrying operative or other treatment altogether, if such be indicated; but let no one be deceived into expecting benefit from the procedures, except in rare instances.

Having briefly examined most of the general physical disorders which are concerned in the causation of insanity, we will now turn our attention to certain functional diseases of the nervous system which, by reason of their localization in the cerebral cortex, are prone to assume a very important part in psychopathology. These are epilepsy, hysteria, and chorea.

Epilepsy.—Epilepsy is almost as common a disease as insanity itself. Asylum physicians, whose experience with epilepsy is limited to cases associated with mental disorder, tend to overestimate the frequency of insanity among epileptics. Thus, it is often stated by them that psychic degeneration is manifested in sixty to eighty per cent. of all epileptics. But the fact is that probably not more than ten to fifteen per cent. of epileptics develop insanity; at the same time the proportion is so large as to show a close relation between this functional cortical malady and mental disorders. When progressive epileptic degeneration occurs, it manifests itself by the following symptoms:

1. Slowness of ideation and articulation.
2. Abnormal irritability of temper.
3. Hypochondriacal depression.
4. Paranoid outbreaks of various kinds.
5. Dementia.

Hysteria.—Hysteria is also a functional neurosis of the cortex, often closely associated with divers psychoses. There is a species of hysterical psychic degeneration, and the neurosis frequently gives a special color to different forms of insanity. The symptoms noted (aside from the peculiar sensory and motor manifestations familiar to us in simple hysteria) on the mental side are as follows:

1. Lack of logical coherence and sequence of thought, but with perfect intelligence. Defects of memory, with rudimentary persecutory and erotic delusions, are encountered frequently.
2. Extreme uncontrolled and morbidly changeable emotions. Profound egotism.
3. Frequent illusions; occasional hallucinations.
4. Conduct and speech are based upon emotional impulsiveness, uncontrolled by ethical considerations of any kind.

Organic Nervous Diseases.—The psychic disorders induced by organic processes in the brain, such as meningitis, tumor, softening, hemorrhage, and the like, are characterized either by symptoms of retardation of functions or by symptoms of irritation, and are due either to pressure or to the indirect influence of the lesion upon the circulation or nerve-centers and tracts. Emotional irritability, hallucinations of the various senses, defects of intelligence reaching to imbecility or idiocy, stuporous conditions—these are common mental manifestations of such processes. Since single localized lesions are apt to produce slight mental changes, any marked intellectual defect or multiform psychic symptoms may be looked upon as suggestive of wide-spread, perhaps multiple, lesions, such as multiple sclerosis, multiple tumors, syphilis, etc. Sometimes true insanities develop in these cases, particularly when there is hereditary instability of nervous organization.

Trauma to the Head.—A blow upon the head is one of the most direct of stresses to which the brain can be subjected. It is not so much the local effect of the injury (which, indeed, would not present psychic symptoms differing materially from those of any other local lesion of the brain such as have just been considered), but the general effect of a commotio cerebri that we are called upon to consider. The syndrome of mental disorders induced by such cause has been well termed by the Germans "commotion insanity." The effect of a violent blow, jar, or jolt to the head must have some influence upon the molecules of the brain as well as upon the encephalon as a mass, must displace and disarrange delicate microscopic structures, such as the cells and fibers. If the blow be insufficient to produce complete loss of consciousness, there will be a dazed, bewildered condition, and the patient will struggle or grope about in a confused way. There may be a loss of memory, more or less extensive, as a result. Naturally, the newest organizations of tissues, being the most fragile, will be the most easily disarranged; hence, with amnesic defects, it will be the most recent acquisitions, or such as cluster about the time of the injury, which will suffer most. The patient will experience strange sensations in his head. The head may feel as if it were going around. Objects seem to move. There is a feeling of being intoxicated or of dizziness. A general hyperesthesia and hyperalgesia are not uncommon, while a hypalgesia is occasionally observed. Among psychic symptoms hallucinations and painful effects are prominent, generally of a terrifying nature. Associations may be so interfered with as to induce difficult ideation, mental confusion, and a genuine primary incoherence. The motor expressions are often characteristic, consisting of catatonic conditions, impulsive acts of violence, and aimless wandering about. In some cases no particular results of the trauma are noted until the lapse of a few hours or days, when suddenly the traumatic psychosis develops. After the psychosis has run an acute course, a condition of chronic insanity or of a secondary dementia may follow. Such a secondary dementia may simulate very closely general paresis, particularly if it be progressive. It can not be said that there are any well-authenticated cases of true general paralysis dependent upon traumatism.

There are not infrequently instances of the creation, by trauma to the head which has induced no direct evil consequences, of an unstable nervous system, of a predisposition upon which other etiological factors may operate later in life.

Insolation probably acts upon the brain in a manner similar to traumatism.

Nervous Exhaustion.—Stresses of various kinds, mental or physical, especially in conjunction with the impairment of the nutrition of the central nervous system, induce an exhaustion upon the basis of which a psychosis may develop. The mental strain may be from overwork, overstudy, insomnia, and the like; the physical from masturbation, sexual excess, hardships. The nutritive impairment is the result of some blood-change or deficiency, such as constitutional anemia, a cachectic state, etc. The physical symptoms of such exhaustion are: slowing of the thought processes, difficulty of recollection, want of ability to concentrate the attention, rapid fatigue on mental exertion, emotional irritability with an undertone of depression, leading often to fully developed insanities, which are designated as asthenic psychoses. The common features of such psychoses are retardation and incoherence of the mental processes, manifested even in the quality of the hallucinations and delusions. But almost any form of insanity may be evolved from this asthenic state of the nervous system, such as mania, neurasthenia, stuporous states, and various types of paranoia.

As Ziehen points out, it is also interesting to observe how any exhausting psychosis may in itself induce this asthenic condition with the characteristic features of an asthenic psychosis, as a result of which we have a secondary type of mental disorder developed upon the basis of the original insanity.

Physiological Factors.—Puberty, the puerperal state, the climacteric, and senility are indirect strains to which the organism is subject, by reason of the more or less profound physiological commotions they arouse in the nervous system—commotions which may well disturb the normal adjustment and equipoise of the thousands of delicate processes going on in the brain, and thus enormously increase its vulnerability to the direct factors which beget insanity.

The curve of psychic morbidity reaches its highest points, corresponding to maximal aggregations of etiological factors in both sexes, at puberty, middle age, puerperal periods, the climacteric, and senility.

Puberty.—From the thirteenth to the twentieth year there are remarkable changes, physical and mental, in the growing individual. These are more noteworthy in the female than in the male sex, for the time is shorter for the change from girlhood to womanhood than from boyhood to manhood. The evolution of the sexual characters and the development of the powers of reproduction induce a stream of innumerable new stimuli from the genital organs to the brain, accompanied by wholly new organic sensations, new associations, and new and powerful emotions. The evolution is rapid, and, as is the case with all rapid development, more or less unstable.

The boy grows fast in body, takes on the aspect of manhood, with a stronger and more rugged frame, a changing voice, a budding beard. His mind is filled with new sensations, emotional, sentimental, amatory, and with changing, fantastic, illusory dreams and imaginings. Even in the normal youth this nascent state, this struggle of the emotions, thoughts, instincts, impulses for new associations and new combinations, may be greatly aggravated in many cases by masturbation, in others by nutritive disorders. If this be so with the normal individual, how much greater must be the stress of puberty to the individual with a constitution vitiated by hereditary taint!

The girl leaps more quickly into her place in life. The physical changes are more rapid in her, and at the same time more varied and noteworthy. It is a time of tumultuous activity of mind and body in an organism which has not the numerous outlets for surplus energy possessed by the other sex.

The psychoses of puberty are various in their expression. They may manifest themselves as a mania, a melancholia, a paranoia, or as an insanity with peculiar color, to which the name hebephrenic modification has been given; so that we speak of a hebephrenic mania, a hebephrenic melancholia, etc.

By the designation hebephrenic is understood the following syndrome: Extraordinarily rapid and paradoxical changes (depressed ideas in the midst of boisterous gaiety, jocularity in the deepest depression), with paradoxical facial expression and paramimia; exalted motor activity (laughing, dancing, grimacing, exhorting after the manner of an orator, often with incomprehensible words and sentences); conduct and action without apparent object, but often with the semblance of desiring to attract attention.

Puerperal State.—Pregnancy, parturition, and lactation diminish the vitality of woman, debilitate and weaken her entire organism, induce a species of physiological commotion in her nervous system, and, in short, bring to bear a strain upon her which is, even under normal conditions, attended by emotional irritability, depression, morbid yearnings, etc. It is not strange, therefore, that the puerperal period in women with unstable nervous systems should often be an exciting factor in the development of psychoses of various kinds. In about ten per cent. of insane women the insanity has its origin at the epoch of reproduction. The majority of these cases are parturitional (seven and a half per cent.), while about a fourth are lactational and a tenth pregnancy cases. It is perhaps true that there are many cases of parturitional psychoses in which the insanity is not so much due to the stress of labor as to possible auto-intoxications from poisonous substances absorbed during the catabolic changes incident to subinvolution of the enlarged uterus. As important factors, too, we must include loss of blood, parametritis, sepsis, mastitis, etc.

There are divers forms of insanity consequent to the puerperal state, such as acute hallucinatory paranoia, melancholia, stuporous insanity, mania, and neurasthenic insanity.

Menopause.—The climacteric, between the ages of forty and fifty, is another epoch of change in woman, a period of involution in its way analogous to the evolution at the age of puberty. There is a physiological commotion in the nervous system at the time of the cessation of ovulation and menstruation, a disequilibration associated, even in normal individuals, with numerous neurotic manifestations, and, in such as have unstable organizations, attended with peril to the mental integrity. Melancholia, simple and hallucinatory paranoia of chronic character, and circular insanity are the forms of psychosis incident to the menopause. About four per cent. of the cases of insanity in women are due to the climacteric.

Senility.—The involution of all the tissues of the body characteristic of especially the seventh decad of life forms also a frequent basis for insanity which depends, in the main, upon the loss of functional activity in the cerebral cortex. Such loss is notable even in normal individuals. The latest acquisitions of the mind are the least stable; hence the conspicuous loss of memory for events of recent occurrence and the tendency to live in the past. The scope of interests, sympathies, and ideas narrows itself down to the individual's immediate physical comfort and needs. While the physiological involution of senility belongs to the seventh decad, in many instances it begins long before this, owing to general debility, endarteritis, etc. Marked changes in the brain of such nature must, therefore, often superinduce veritable psychoses in individuals predisposed to mental disorder by heredity or by antecedent physical or psychic stresses.

Senile insanities manifest themselves in many forms,—melancholia, mania, incoherent paranoia, hallucinatory paranoia, dementia,—but, of course, modified from the common types by the weakening of the cortical functions previously described. Vertiginous seizures, slight pareses, dreadful hallucinations, and primary anxious conditions are often observed in all of these forms.

The hallucinations appear in senile forms in psychoses which ordinarily run their course without them, and the anxious states in ordinarily non-affective insanities.

Moral Causes.—About twenty-four per cent. of all cases of insanity are ascribed to moral causes, among which are classed domestic troubles, grief over death of friends, business worries, anger, religious excitement, love affairs, fright, and nervous shock. The percentage is greater in women than in men. It is doubtful if any emotion alone can overcome the stability of the normal nervous system; hence it is in the fragile, nervous constitutions of individuals tainted by heredity that extreme emotions are wont to exert their malign influence.

The uncertain equilibrium of the highest nerve-centers in these cases is all too readily overcome by the tumultuous wave of an intense emotional impression. Possibly, the results depend upon disturbance of the vascular innervation. Ordinarily, the greater and more sudden the emotion, the greater the liability of the badly poised brain to succumb; but, like the drops that wear away the stone, an emotion of less intensity may, by long continuance, produce equally disastrous consequences. Some acute psychoses may be suddenly developed by fright, or a transitory emotional insanity for a few hours or for a few days in duration. Among the symptoms are mutism, or incoherence, confusion, isolated hallucinations, delusions, with impulses to violence and aimless wandering, followed later by complete, or nearly complete, amnesia.

The more slowly working affects, like sorrow and worry, often aid in the evolution of melancholia, paralytic dementia, or acute or chronic paranoia.

Imitation.—The so-called psychic infection never influences normal individuals who are brought into contact with the insane. Physicians, attendants, and others who have to do with the insane are never affected, except when morbid heredity and mental and physical overwork combine to prepare the soil for the development of a psychosis. The writer recalls but one instance of an attendant being mentally unbalanced during her service. She was neurotic by constitution and cut her throat a few days after one of her patients had committed suicide in the same manner. But there are not infrequent instances of communicated insanity among members of a family.

The simultaneous development of insanity in two or more persons associated together, or the imposition of delusions gradually arising in the mind of one upon the impressionable intellect of a second, third, or of many persons, has been described under the names *Folie à Deux*, *Folie Simultanée*, *Reciprocal Insanity*, *Folie Imposée*, etc. There are several factors which govern the evolution of such insanities. In both forms a degenerative soil is usually required for the proper germination and growth of morbid ideas. In the simultaneous variety there must be, in addition to predisposition, that similarity of intellectual substrata which we find particularly in persons who are blood-relations or who are intimately joined together by mutual like and dislike; hence it is that brothers or sisters most frequently manifest simultaneous insanity. Take two healthy children of one family and bring them up far apart, yet there will be innumerable physical resemblances between them, and many peculiarities in their character and conduct which prove them to be consanguineous; if a hereditary instability of nerve-cells had been implanted in them, there would be a tendency to a similar form of dissolution, even if they remained apart. How much greater would this be in two persons so intimately associated as sisters, for instance. In children the study of unconscious imitation is one of great interest. Who has not observed the identity of intonation of phrases, of gesture, of laughter, of many facial expressions, of certain habits, in children either related or brought up together? Such unconscious imitation, as is well known, may lead in children to the contraction of certain nervous and even mental diseases. The contagious quality of emotions is well established. An explosion of laughter will call up smiles on even melancholy faces in a crowd. A pathetic scene on a stage will bring tears and depress the oral angles in a large audience. The unconscious imitation of gestures, such as bowing, often seen in adults, is in a milder degree such mimicry of motion as is observed in dancing mania.

Another element in the imposition of insanity by one upon another is the quality of the morbid mind-product. If a delusion, it must have an air of probability to the person receiving it, and must be gradually developed and imposed. It is because suspicion is inherent in the nature of most persons, because suspicion can wear so much probability of truth, that persecutory delusions are by far the most frequently adopted by others. Credulity is an important factor in the imposition of insane delusions upon others. It was the ready credulity of large numbers of persons, especially as regards religious subjects, that in the past led hundreds of thousands of people to adopt with faith the delusions of paranoiacs like John of Leyden, John Thom of Canterbury, Joan of Arc, Richard Brothers, Joanna Southcott, John Brown, and many others, and actually to sacrifice their lives upon the altar of their beliefs. Though these delusions emanate from an insane person, their acceptance by others does not, of course, necessarily imply insanity in the latter, for delusions of this character have their support in the superstitions of many and in ignorance concerning supernatural matters. A persecutory delusion might be imposed by an insane person upon an intimate associate, and yet the latter need not, of necessity, be insane; but when the exposed individual adopts the delusions, regulates his conduct upon them, allows them to become rooted in his mind, even begins to share the hallucinations of his friend, there is, of course, actual aberration of mind present. Several cases of *folie à deux* have come under my observation. One case was that of two sisters, aged about fifty to fifty-five, Irish, washerwomen, who, living alone in a tumble-down shanty, were often tormented by boys throwing stones at the house at night, and otherwise teasing them. They finally developed persecutory delusions with hallucinations, and both were very much alike. They became so violent in their demonstrations that ere long both were taken to the asylum, where I took charge of them. They were separated, the result being that one became rapidly demented and the other became a quiet worker, with fixed persecutory ideas and auditory hallucinations.

Another pair of sisters, colored, between forty and fifty years of age, were similarly affected. For ten years one sister had been a paranoiac, with delusions of persecution by means of electricity, which was at all times, night and day, hurled through her body by a vast organization of conspirators. She had hallucinations of hearing. The sisters had not lived together until within six months of my seeing them, the sane sister having recently become a widow. The sane sister gradually adopted the delusions of the insane one, and probably the hallucinations. She believed her sister to be persecuted by an organized band of conspirators with electrical appliances.

A third case was that of a husband and wife, who both became typical cases of melancholia, with, of course, similar delusions, one shortly after the other. Such a case as this might be called a coincidence, and not an imposed insanity. Probably grief over the insanity of the husband was one factor in developing that of the wife, but unconscious emotional imitation between two persons united by special bonds of sympathy was undoubtedly another element.

A fourth example I detailed fully some years ago in the "Alienist and Neurologist" ("Paranoia in Two Sisters," January, 1890):

C. K. and H. K. were respectively thirty-six and forty-two years of age, teachers of music and singers by occupation, of German parentage, and had both been insane for some ten years. Their mother was a case of paranoia, with fixed delusions of an exalted religious nature. She believed herself to be the mother of God. She was never in an asylum, but lived at home until her death. While insane she gave birth to the younger of the two sisters, C. K.

One of them wrote for me an autobiographical sketch, and the other some twenty-five letters, upon which the following facts in their identical clinical history are based:

The instigators of the conspiracy against them are chiefly their uncle, brother-in-law, and sister-in-law, and a brother has also been inveigled into it. By them are employed numerous detectives, expert chemists, and handicraftsmen, and, as they have privately hinted to me, also many lawyers. Openings are made in their rooms in spite of all they can do for the insufflation of noxious gases, smoke, camphorous, chloral, and chloroform vapors; and by some unseen agency substances are thrown at them which produce painful cutaneous eruptions. Their food and water and heating apparatus are tampered with for the introduction of poisons or to produce serious illness. They hear the mechanics at work upon the floors, walls, ceilings, and the voices of the detectives (hallucinations of hearing). Their food has a peculiar taste (hallucinations of taste). Most prominent of all are the singular odors of the room, of fruit and flowers sent them, of the water (hallucinations of smell). Sometimes they are black in the morning when they look in the mirror (illusion of sight). They are subject to remarkable, generally painful, sensations in their bodies (hallucinations and illusions of cutaneous sensibility).

They hint of imaginary property in Germany, out of which they are being defrauded by relatives. For ten or twelve years they have been driven from one place to another in Brooklyn and New York by their pursuers. As yet they have sought only escape and protection from persecution; they have very rarely manifested anger by pounding the floor when hearing the mechanics at work or by complaint to the landladies, and have not been brought to bay to a condition in which they might turn upon the actual instigators of the conspiracy and do them bodily harm. They have been on the point of a visit to police headquarters to make declaration against their enemies.

From what I can learn of their history in youth the two girls differed from others of their age in a slight degree, some trifling eccentricities and some overweening self-consciousness constituting this difference. They have always been closely united—living together, sleeping together, having the same affinities, talents, pleasures, and pursuits. The development of suspicions and delusions of persecution had been so gradual that it did not become evident to others that they were actually insane until a comparatively recent period. When I first saw them in my office, they came heavily veiled, and, upon removing their veils, their faces were patched all over with small square pieces of cloth, covering sores. These were only an ordinary acne, made much worse by picking, by wearing wet cloths on their faces all night for the purpose of preventing poisonous vapors from entering their lungs, and by the removal of the strongly adhering pieces of linen from the bleeding surfaces. They healed up rapidly when I had prevailed upon them to make use of ung. zinci ox. freely. The face of the younger is particularly characteristic of a degenerate type, one of its features being a disagreeable prognathism.

Some of the skull diameters were pathological in character. One of the sisters died in convulsions from unknown cause, which the other sister still attributes to poison. The living sister still moves about from one part of the city to another, cherishing the paranoiac delusions, but supporting herself in part by teaching music.

CHAPTER III.

GENERAL SYMPTOMATOLOGY OF INSANITY.

EVERY psychic phenomenon is accompanied by a material process in the cortex of the brain. There is no insanity without disease of the cortex. The material disorder of the cortex is diffuse and partly organic, but mostly functional in character. We term it functional, for thus far our pathologico-anatomical and clinical studies have failed to reveal any definite material basis for the majority of psychoses.

The progress made of late years in the study of physiological psychology has illuminated many obscure features of morbid psychology, and has put us in a position to better examine and classify the symptoms of insanity.

There are material processes in the central nervous system unaccompanied by any parallel psychic process. The reflexes and automatic acts are examples. In these phenomena we observe a stimulus, a sensation, a movement. Movement paralleled by a psychic process becomes action. We sometimes speak of conscious voluntary action. Action differs from simple movement in being accompanied by intercurrent images—memory-pictures of former stimuli. A peripheral stimulus excites a cortical center, and is not carried at once to the motor region, but travels first by association fibers to the area in which are stored up residua of former similar stimulations, and later to the motor region. These residua of memory-pictures or ideas may be complex, constitute a series, have many associations, and hence we designate them as an idea-association. Action, therefore, consists of the series: stimulus, sensation, idea-association, movement. The various ideas thus excited tend to different motor expressions, so that the resulting movement or action will depend directly upon the strength of ideas. The stronger ones conquer. Ziehen, whose clear explication of the mental problems of psychiatry the writer closely follows,[1] has well described idea-association as the play or battle of motives. He gives the following example of the physical and psychic processes just described:

I see a rose in a strange garden (stimulus and sensation). A long series of ideas is aroused by the stimulus and the visual sensation of the flower (idea-association). For instance, the memory of the rose's fragrance comes to mind, then I think how well it would look in my room, that it is the property of another, that plucking it would be punishable, and so on. Only after the whole series of presentations has passed before the mind does action follow, and whether I pluck the flower or go my way without it will depend upon the strength and intensity of the conquering idea.

Every psychic process must be regarded upon the basis of such a scheme, and as accompanied by its material parallel (progress from the sensory cells to the idea-cells, and from these to motor-cells by means of association-fibers).

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[1] "Psychiatrie," Th. Ziehen, Berlin, 1894.

Sometimes the idea of movement (memories of former sensations of movement) comes before the movement in the series just described, but generally the movement is perceived after it has taken place by means of the sensation of the movement.

There are really but two psychological elements—viz., sensation and idea. The only process connected with these elements is the idea-association. Their product is action. The so-called mental powers of old-time psychology do not exist. The assumption of a special power of will dominating the idea-association and voluntarily determining this or that movement is particularly superfluous and misleading. The assumption of a special power of apperception which turns its "attention" voluntarily upon this or that idea or sensation to determine the course of the idea-association is equally superfluous.

The presentations or ideas rather follow one another according to laws without intervention of any especial voluntary power of the mind, and the final movement or action is the necessary result of association of these presentations or ideas. Finally, there exists no particular faculty of feeling, for exact investigation demonstrates that our feelings of what is agreeable and what is distasteful, of pleasure and pain, appear never in an isolated state, but always combined with sensation and idea as attributes or properties.[1]

Following Ziehen in these particulars, we shall study pathological psychological processes on the basis of the scheme just described, and in each case investigate, first, disorders of sensation; then, disorders of the memory-pictures, presentations, or ideas; then, again, disturbances of the idea-association; and, finally, the influence of these disturbances upon the actions or conduct of the patient.

DISORDERS OF SENSATION.

Sensation is the first element in the psychic process. It is determined by some external stimulus affecting any sensory nerve. Every sensation has four important attributes—viz., *quality*, *intensity*, *tone* (the accompanying feeling of pleasure or pain), and *space-projection*. We are not especially concerned with the last in morbid psychology.

Qualitative Disorders of Sensation.—The two important classes of qualitative disorders of sensation are *hallucinations* (in which we have sensation without external stimulus) and *illusions* (in which we have the external stimulus, but a transformed or perverted sensation). An external stimulus to a peripheral nerve is carried to the cortex, where it acts as a secondary stimulus in exciting sensation.

Hallucinations.—A hallucination is a sensory impression without external stimulus. It is often also defined as a perception without an object. The patient hears voices where all is silent, sees forms and figures in empty space.

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[1] "Leitfaden der physiologischen Psychologie," von Th. Ziehen, Jena, 1893.

Hallucinations of sight are very common, and vary from the simplest sparks, lights, shimmers, flames, spots, threads, clouds, and shadows to the most complicated groups of persons and landscapes with perfect details. Sometimes they are colorless, like silhouettes; sometimes radiant and fantastic with color. Sometimes they are flat, like pictures; sometimes plastic. Ordinarily, the forms and objects observed are of natural size, but occasionally they are gigantic or diminutive. They may appear close at hand or far away. They may be quiet or full of movement, like the zoöscopic hallucinations of alcoholism. Rarely, real objects are doubled or multiplied (hallucinatory diplopia and polyopia). Real objects are sometimes concealed by the hallucinations, sometimes merely diaphanously veiled. Hallucinations may fill the whole field of vision or appear in homonymous half-fields, as in the hemiopic hallucinations described by the writer in cases of insanity and of homonymous hemianopsia.

Hallucinations of hearing are also extremely frequent, and vary from simple sounds, tinnitus aurium, rushing, roaring, whispering, tinkling, to complicated music and words and sentences. These last may be in natural tone or deep-voiced, whispered or loud; may be the voice of one or many persons; may be pronounced in various languages; may be single words or long orations; may seem near at hand or far removed; and may be heard in one ear, though usually in both. Not infrequently the voice seems to the patient so near that it appears to be in his head or body.

Hallucinations of common cutaneous sensibility may appear anywhere in the skin or in mucous membranes in the form of electric shocks, pricking, tingling, blows, caresses, sensations of heat or cold, indignities to the sexual organs (feeling of cohabitation), etc.

Hallucinations of smell are very common. The patients perceive odors of chloroform, sulphur, noxious gases, smoke, filth, or, on the other hand, the smell of perfumes and flowers.

Hallucinations of taste are so generally combined with those of smell, because of the close physiological relation of the two senses, that true hallucinations of the primary elements of taste (salt, sweet, bitter, and sour) are uncommonly rare. A hallucination of a bitter taste is the most frequent. On the other hand, the combined hallucination of taste and smell (as of blood, filth, etc.) is rather common.

Hallucinations of organic sensation are not rare. The patients complain of peculiar or extraordinary feelings in various organs, such as malposition, gnawing, cutting, pain, etc.

Hallucinations of active or passive movement of the body or its parts depend probably chiefly upon disorders of joint sensibility. The patients feel themselves lifted in the air, floating, the limbs moved actively, the head turned to one side; or, the sensation of movement of the muscles required for speech may give rise to the hallucination of having spoken a word or sentence.

Various hallucinations are often associated in such a manner as to render the hallucinated objects still more natural and deceptive, though more frequently they are not thus commingled. Thus, visionary figures may speak or be dumb, and the fancied voices may come from visually projected or from unseen persons. Sometimes vision, hearing, and cutaneous sensation may be combined to give reality to the object. Combinations of others are also met with, and, indeed, these mixed hallucinations are common and multiform.

As regards the development of hallucinations, some are doubtless peripheral, but the majority are central in their origin. Disorders of the eye, ear, nasal cavity, mouth, mucous membranes, skin, and viscera may give rise to hallucinations, though they are more commonly the cause of illusions. Hallucinations are never new creations, but are made up of memory-pictures stored up in the cortex; these may, however, make their appearance in new combinations. The congenitally blind never have visual hallucinations; the congenitally deaf never auditory hallucinations, though they are noted in acquired blindness and deafness.

Hallucinations are usually of two kinds—those which have to do with the ideas presented in the mind at the time of their manifestations, and those which are concerned with latent memory-pictures. The former are more common, but both may be observed in the same patient. The first kind are those which the patient describes as visions which picture his very ideas, and voices which read off his thoughts as fast as they can come into his mind—indeed, often apparently before he thinks them. The second class of hallucinations often astounds the patient by association with things long past and quite forgotten.

We are taught by physiological psychology that a stimulus to the eye arouses a sensation in the occipital lobe, to the ear a sensation in the temporal lobe, and so on, the sensation further exciting an image which remains as a memory-impression. All normal sensations, then, depend upon the series stimulus, sensation, memory-picture, or idea. Now, hallucinations are always cortical, as regards localization, and depend upon a reversal of the normal course just described, and without the stimulus. The memory-image is excited and then excites the sensation. A certain irritability of these centers will be induced, undoubtedly, by morbid processes in the peripheral nerves or their terminations, such as entoptic or entotic processes, which will render them all the more excitable, since external stimulus is not then altogether wanting. Finding such,—and we should always investigate carefully for a peripheral physical basis,—the dividing-line between hallucinations and illusions becomes less distinct. Naturally, the normal mind recognizes the real nature of muscæ volitantes, tinnitus aurium, neuralgic pains, etc., and it is only the abnormal mind which employs them as material for illusions and hallucinations.

In the examination of a patient we must determine the presence of hallucinations and the effect of their presence on idea-association. One must not mistake actual occurrences described by the patient, nor the events of dreams confused by him with events of waking, nor ordinary illusions for hallucinations. There is danger, too, of overlooking their presence. Patients conceal them, conscious that the hallucinations are morbid, or knowing that they will be looked upon as such, but will often write about them or tell of them to other patients if opportunity be given. Very often the physician is enabled to recognize their existence from the expression and conduct of the patient.

As regards the influence of hallucinations upon the course of idea-association, the most important question is whether they are regarded by the patient as real sensations or not. He treats them as actual phenomena, as if they were normal sensations, or he distinguishes them from his ordinary sensations as peculiar, novel, and possibly inspired by supernatural agencies; or he is really conscious of their morbidity, but may believe them to be induced by enemies by means of poison. If the hallucinations are faint and transitory, the patient may not be much influenced by them; if they are marked and persistent for a long period, he ultimately loses his critical faculty and comes to believe in their reality. Such being the case, his thought and conduct are bound to be influenced by them, and more powerfully influenced than by normal sensations, or by any reasonable consideration or argument. Hallucinations either inhibit (hallucinatory stupor) or retard (hallucinatory confusion) the idea-association; or they induce direct intrinsic delusions (as when a voice cries "Thou art God," and the patient immediately believes himself to be God). The actions and conduct of a patient are very much influenced, and in multiform ways, by hallucinations. He has the expression of listening, or stares apparently at nothing. He closes his ears, covers his eyes or head, closes up cracks and openings, or listens at the window or keyholes. He refuses or spits out his food. He holds his nose, or suddenly closes the window to prevent the entrance of noxious gases. He turns his head, runs, shouts, lifts his arm quickly, or takes peculiar attitudes, acting upon a hallucination of muscular sense (imperative movements, imperative speech, imperative attitudes). The imperative attitudes may be very persistent and long-continued, and are then called catatonic. Hallucinations often lead to imperative acts which may be of a violent nature. If hallucinations are innumerable, very changeable, and intense, the patient is affected by so-called hallucinatory agitation.

Hallucinations are so extremely rare under normal conditions that they are to be considered as almost always pathological. Illusions are rather common in the normal mind. True hallucinations may occur in apparently normal individuals, but examination will show that such persons are neurotic by heredity, and that some stress of mind or body has induced this psychopathic phenomenon. This is particularly true in childhood.

Outside of the psychoses, hallucinations are met with in toxic states, fevers, cachectic conditions, sun-stroke, and some of the neuroses (epilepsy, chorea, hysteria). A hallucination of any sense may be the aura of an epileptic attack; sometimes, when visual, it may be hemiopic.

Hallucinations are the chief symptom of one form of paranoia. Other psychoses, such as mania and melancholia, manifest them only exceptionally; while still others, like senile and paretic dementia, present hallucinations, it is true, but not in such prominence as to make them a characteristic symptom. Visual hallucinations are more common in acute than in chronic psychoses, and they are seldom independent of hallucinations of feeling and hearing. Auditory hallucinations, on the other hand, are more characteristic of chronic types of mental disorder, and are often observed alone.

The close union of the auditory center with the motor speech center gives a peculiar interest to hallucinations of hearing. From infancy man is trained to think to a great extent in word-images or speech-images, and thinking is, therefore, nearly always associated with some stimulation of the speech-muscle centers in the brain. Therefore, hallucinatory irritation in the auditory area of the brain causes synchronous irradiation to the motor speech center, and words and sentences are heard by the hallucinant as if projected into the external world, or into some part of the patient's body (head, throat, chest, stomach, or even extremities). The stimulation of the speech muscles, however feeble, may be sufficiently strong to induce recurrent sensations of movement in them, which leads the patient to imagine that his thoughts are being read off internally by the voice, and sometimes repeated apparently before the thought has fully developed in his brain.

Illusions.—An illusion is a false perception. There is a stimulus but a perverted sensation, a wrong interpretation. The sensation corresponds only in part to the stimulus. A patient hears the rain falling, but perceives it as music; he sees the bedpost, but imagines it a ghost.

Visual illusions exhibit a transformation of form, or color, or both. This is often favored by indistinctness of outline, as when it is half dark or there is a shimmering, flickering light. But often clear outlines are transformed. The patient may see the familiar faces about him changed into those of strangers, transformed by grimaces, or deathly pale. A sharp distinction between illusory transformation and actual hallucination is often difficult to draw. It is peculiar to illusions that they not infrequently present objects as distorted and diminished or increased in size. This is especially true among epileptics. When this is noted with all objects, it often depends upon entoptic disorders. Thus, metamorphopsia may arise from astigmatism and retinal disease, micropsia from paresis of accommodation, and macropsia from spasm of accommodation. When this is not the cause, perverted association of the sensation, with disordered muscular sense, may play a rôle. Sometimes, though rarely, the illusion may consist of a perversion of color analogous, for instance, to the yellow appearance of objects in santonin-poisoning (due to violet blindness induced by the poison) or to red vision (erythropsia induced by fatigue of the retina for the short-waved rays of the violet side of the spectrum).

Illusions of hearing consist mostly of the construction of words out of inarticulate sounds, or of the misinterpretation of the words or sentences spoken in the patient's hearing. He may transform them into mocking, indecent, derogatory, or flattering words.

Illusions of common sensibility are, perhaps, more important in insanity than hallucinations of this sense. But they are difficult to study and establish. It is probable that the sandy, earthy taste of food often complained of by patients is more an illusion of touch than of taste.

Illusions of smell and taste are, in the main, unpleasant in character and are more common than hallucinations of these senses.

Illusions of organic sensation are frequently noted, and consist of such sensory metamorphoses, for instance, as the mistaking of intestinal motions for pregnancy, and the feeling of diminution or increase in size of various organs (particularly noticeable in epilepsy and paresis).

Illusions of muscular sense or of movement are rare.

Illusions, like hallucinations, may form their material from the concepts at the moment in consciousness, or from latent memory-pictures.

The theory of the cause of illusions is analogous to that of hallucinations. They arise from a pathological recurrent influence of the excited memory-picture cells upon the sensory cells. The difference lies in the association, also, of an actual external stimulus which undergoes transformation.

Illusions are much more common than hallucinations, and are not seldom met with in normal persons. Often they are difficult to distinguish from one another. Sometimes it is impossible to differentiate true illusions from so-called illusionary judgments, in which we are concerned not so much with a transformation of sensation, as with an erroneous judgment of the character of a normal sensation.

Illusions are noted in all forms of psychoses, especially in acute forms. They are particularly noteworthy in the hallucinatory form of paranoia.

Disorders of Intensity of Sensation.—These consist of hypesthesias, anesthesias, and hyperesthesias. Hypesthesias and anesthesias are observed in various psychoses which are complicated by such disorders as hysteria, chorea, multiple neuritis, tabes, focal cerebral lesions, etc. Hyperesthesia is also encountered in complicating disorders, such as hysteria, tubercular meningitis, neurasthenia; but is also often noted in the prodromal stages of many acute psychoses. It is especially remarkable in the insanities of childhood. A valuable objective sign of hyperesthesia is exaggeration of the superficial reflexes.

Disorders of Sensory Tone.—Agreeable or disagreeable feeling, associated with sensation, is described as sensory tone. Sensory tone may be perverted in insanity so that, for instance, fragrance is perceived as unpleasant, dissonance as pleasant, and vice versâ. One notes such perversions in the slight psychopathic conditions of pregnancy in the form of capricious tastes and appetites. Homosexual perversion is a form of this disorder manifested in the domain of sexual sensation. Pathological disorders of the intensity of sensory tone consist of hypalgesia and analgesia, hyperalgesia, hyphedonia, and hyperhedonia.

The hypalgesias are noted in hysteria, tabes, congenital and acquired mental deficiency, and in severe hallucinatory confusion.

Hyperalgesia is observed under the same circumstances as hyperesthesia. It is most often seen in hysterical and neurasthenic insanities, and almost exceptionally at certain points (such as the supraorbital, infraorbital, mental, Valleix, iliac, intercostal, mammary, vertebral, and cranial suture points) pressure elicits pain. The pain of hunger, which leads in many psychoses to pathological hunger (bulimia) belongs in this category.

Hyphedonia is a morbid diminution of the feeling of pleasure in any sensory perception. It is more important in the domain of sexual sensations than in others, where it may reach the degree of anhedonia. Sexual anhedonia is not uncommonly developed on the basis of a serious hereditary degeneracy, and is frequent, too, in organic disease of the central nervous system (tabes and paresis), as well as in toxic conditions (alcohol, cocain, morphin). Hyphedonia, in connection with hunger sensations, may reach the state of complete psychic anorexia in some insanities.

Hyperhedonia is a morbid increase of positive sensory tone (agreeability of sensation), and is noted most often in relation to sexual sensations.

Disorders of Memory-pictures or Ideas.—Every stimulus in arousing a sensation in the cerebral cortex leaves some material vestige or impression, which remains as a latent memory-image or picture, latent presentation, or idea. Countless numbers of memory-pictures left by innumerable sensations of all kinds are stored away as a material deposit in the brain-cortex. These are rearoused either by the same or a similar stimulus, or excited through the stimulus of some idea-association. Only a few of the millions of memory-pictures are awakened to life at any one moment; all of the others remain latent. The general concept of any particular object is made up of the association of many centers in the brain, some of which are far apart, such as the smell, feel, taste, color, sound, and name of the object. The relation of this object to others of its kind is present in other associations, and these again in others, so that the material basis of an idea must be a perfect network of association fibers; and all of this labyrinth is connected with the complex series of language-centers, but particularly with the motor and auditory speech-centers, which are trained up from earliest infancy to associate the spoken word with the concrete conception. A word, therefore, expresses, like an algebraic x, y, or z, some very intricate and complicated formula. Take words like "home," "right," "wrong," and so on, and think what a countless number of associated memory-pictures each one must represent! Words are simply convenient abbreviations which render more easy the use of concepts in idea-associations.

We distinguish in every idea four cardinal properties: (1) The contents or meaning; (2) distinctness; (3) associated affects; (4) energy or intensity.

The pathological disturbances of ideas may be studied under the headings of disorders of their evolution, durability, concomitant affects and associations.

Defective Evolution of Ideas.—The number of concepts stored up in the brain varies enormously under normal conditions with individuals and races. In morbid psychology we find the number of ideas extremely small among congenital defectives, such as the idiot, the imbecile, and the feeble-minded. The idiot may preserve rudimentary memory-pictures of the simplest things, such as food and eating, light, darkness, clothing, but without speech associations; he will have none of other persons or other objects about him. In the imbecile the concepts are more numerous and may be known by name; he recognizes persons and objects, distinguishes simple colors with difficulty, may have number concepts as high as ten; he has a few concrete ideas, but, as a rule, no abstract ideas. The feeble-minded has a larger number of memory-pictures, may have abstract ideas, recognizes the significance of likeness and similarity, and may use the words God, right, wrong, etc., but in reality be unable to tell the meaning of such complex, abstract conceptions. It is necessary, therefore, to avoid concluding that the idea is present because the word is spoken by such a patient, for it is particularly characteristic of the congenitally feeble-minded to be apt with words while deficient in grasp of their meaning.

Disorders in Durability of Memory-pictures.—The forgetting of a memory-picture, when the stimulus and sensation producing it are not repeated, may be considered to be due to its gradual erasure by the influence of the nutritive processes which affect the cortical ganglion-cells equally with all the elements and tissues of the body. This physiological destruction of the memory-picture is always very slow, but by pathological processes may be rendered enormously rapid. The destruction may be diffuse or limited to one sensory sphere (for example, apraxia, where the sensory ideas of objects are lost, though the sensory apparatus may be intact; mind-blindness, word-blindness, mind-deafness, word-deafness, etc.). But these limited defects of memory-pictures are due to focal lesions in the brain, and do not concern the alienist so much as the diffuse destruction of ideas, although it is true that the latter may sometimes be a sequel to a circumscribed lesion, and, on the other hand, that the diffuse disorder may, as in general paralysis, sometimes affect one region more than another. A loss of concrete ideas, such as general concepts of relationship, etc., which are represented by a wide-spread association network in the whole cortex, can only be caused by a diffuse, far-reaching disturbance. We see examples of such loss in the acquired dementias of paresis, epilepsy, and senility, dementias secondary to acute psychoses, and dementias due to toxic agents. It is natural that the latest memory acquisitions should be lost first, and the older memories successively later, in direct proportions to their age, according to a certain "law of regression," as Ribot terms it. This is to be explained by the want of permanence and stability in the newest arrangement or concatenation of protoplasmic molecules and ganglion-cells. The older impressions have become more fixed and durable.

Since an experience leaves behind not alone a single memory-picture, but a whole series arranged in chronological order, we may, as in amnesias, find pathological states in which there are losses of such series of ideas during a definite period of time. The so-called subconscious or unconscious states are examples of this phenomenon. They are observed in epilepsy, intoxications, hysteria, narcolepsy, hypnotism, somnambulism, injuries to the head, and in transitory insanity.

Affective Disorders.—Pleasurable or disagreeable feelings accompany ideas, just as they do common sensations; so that there is an intellectual affective tone analogous to sensory tone. If two ideas be presented simultaneously, and if one of these have a stronger emotional quality than the other, the tone of this will be irradiated to the other. Ziehen, in describing irradiation, gives the following example: "If I have met with an accident in any place, afterward not only is the memory of the injury accompanied by an unpleasant feeling, but the memory of the place is likewise mingled with a disagreeable affect. Furthermore, when I again see the spot where the accident occurred, I may feel again the sensation of the injury, accompanied by its unpleasant sensory tone." Here the memory-picture arouses the sensory tone of the sensation experienced. This is termed reflected tone. The most important consequence of the laws of emotional irradiation and reflection is that if in a certain period of time one or several sensations and ideas have a strong and similar emotional tone, all other sensations and ideas presented to the mind during the same period of time will be colored by the tone of the former. Such irradiation creates our moods, which are hence the abstract or summary of the similar emotional tones of the ideas and sensations experienced within any definite period of time.

Moods and emotions influence strongly the flow of our ideas, and, as a consequence, our actions. Depressed moods or affects inhibit, while exalted affects increase the flow of ideas, and likewise the resultant actions. Depressed affects are more durable and persistent than exalted affects. The latter subside rapidly. The more complicated ideas, such as justice, honor, law, family, patriotism, etc., are accompanied by a specific affect or tone which we designate as ethical feeling. Ethical feeling is the result of numerous irradiations, which the single idea acquires from all of the ideas associated with it; and the sum of the ethical feelings of an individual gives him his character (Ziehen).

In morbid psychology we classify changes in the affects as pathological depression, exaltation, irritability, apathy, and mutability.

Depression.—Depression is observed in many forms of insanity, particularly as a prodrome, but is characteristic of the melancholy types. It is a very common prodrome of acute mania, and a long period of morbid depression is frequently noted as an antecedent in general paresis. It is observed in neurasthenia, in hypochondriasis, and not seldom as an interlude in any psychosis. It is the cardinal symptom of melancholia. Depression is a normal consequence or accompaniment of sorrowful or dreadful hallucinations and ideas, and is, under such conditions, termed secondary. It is primary depression with which we are more concerned in insanity—a depression not at all or but slightly motived by such hallucinations and ideas as we have just described, but a mood which takes possession of the mind of the patient and gives its own original color to every thought arising in his mind and to every external object presented to his consciousness. Past, present, and future are alike under the shadow of this mood. When mild in degree, the patient feels only an inexplicable sadness—a certain restlessness or state of worry; but when extreme, this general mood of sadness becomes a condition of pathological anxiety—a mixed feeling of grief and dread, often accompanied by a feeling of suffocation or pain about the heart, and, therefore, frequently designated as "precordial anxiety" or "precordial fright." When primary depression is present, the patient feels the change in his mental condition, observes that he no longer is cheered by the usual pleasant events of his daily life, that these rather intensify his misery. The affection and sympathy of his friends and family either awaken no response in his own breast when he tends to believe that he has lost all natural feeling, or they may awaken suspicion, dislike, and distrust. The inhibition of the flow of thought restricts his ideas to himself and to the somber contents of his mind. He is not easily distracted from such contemplation, and answers questions, if at all, very slowly and with great difficulty. Nearly all cases with morbid depression complain of disorders of visceral sensibility, from a slight sense of constriction at the throat to precordial distress, from a general feeling of illness and uneasiness to a feeling of extreme and general restlessness. No doubt depression influences often the entire musculature of the body, so that the patient wrings his hands, picks his fingers or head, walks up and down, is extremely agitated, goes into a condition of catalepsy or catatonia, or, on the other hand, remains absolutely immobile and requires the service of others for every movement. The muscles of the peripheral arteries contract and increase the frequency of the heart's action. The constriction of the throat is probably an actual contraction of the esophageal muscles. Precordial anxiety is most likely due to vasomotor disturbance in the vessels of the heart. The constipation so frequent in depressed conditions depends doubtless upon retardation of peristalsis. Thus we observe in one case motor inhibition, in another motor excitement, and in some alternations between the two.

In seeking to explain the mood of sadness and uneasiness which he feels, the patient tends to develop delusions. He invokes the first ideas which would naturally come to him under such circumstances. He seeks in his past life for some sin, the commission of which may have brought this punishment. He magnifies some trivial error in his youth into an unpardonable sin. Or he comes to think that poverty stares him in the face, or that he can never recover from an incurable illness which has taken possession of him. Occasionally, a persecutory delusion is evolved from a primary depression.

Exaltation.—Exaltation is occasionally noted as an intercurrent symptom in any psychosis. It sometimes alternates with depression, forming a constant cycle, as in circular insanity, and sometimes it presents itself during convalescence from melancholia as a reactive phenomenon. In the majority of cases of general paresis a period of exaltation develops. In maniacal states, however, it is observed as a cardinal symptom. As with depression, we distinguish a secondary exaltation consequent upon agreeable hallucinations and ideas, and a primary or unmotived exaltation. In exalted moods the somesthetic sensations are pleasurable and give rise to feelings of perfect health, strength, and vitality. The stream of ideas is hastened, and as a result the patient becomes, according to the degree of exaltation, talkative and garrulous, or exhibits a veritable logorrhea,—a constant, rapid flow of words,—which may often assume a rhyming, singing, or oratorical character, with marked incoherence. The rapid stream of presentations is paralleled in the motor sphere by increased muscular activity, varying from busy occupation with nothing to gesticulating, grimacing, and dancing, and to the wildest and most violent motor excitement.

Primary exaltation frequently gives rise to delusions of a grandiose character, though these are unstable and fleeting, corresponding to the rapidity of change in the contents of consciousness. But the feeling of well-being and of egotism which makes up the fundamental mood of the exalted patient leads him to be extremely impatient of any restraint of his activities; and, in consequence of this, the reactive feeling of aggressive anger and fury is easily aroused, leading to acts of violence and destruction.

Irritability.—Irritability is a condition which has to do chiefly with the affects of anger and rage. While observed in association with exaltation, as just noted, and among the prodromata of various insanities, it is particularly characteristic as a primary emotional state of congenital and acquired mental weakness, neurasthenic insanity, and the epileptic psychoses. In the latter it not infrequently becomes a true *furor epilepticus.* Irritability is occasionally noted in the convalescence from acute insanities, sometimes conjoined with a peculiar tearfulness, a lacrymose irritability. While most of the affects of both depression and exaltation are concerned with the ego, the affect of anger differs markedly from these in that it has to do with persons or objects outside of one's self. At the same time anger is a depressed emotion, but with certain peculiarities. In its influence upon the flow of ideas and upon action it first retards or inhibits, but finally, by an accumulation of stimuli, induces a sudden motor explosion, which may vary from simple aggressiveness to the most uncontrollable fury. Abbreviation of the usual play of motives is characteristic of the motor explosions of anger and fury. The sensory stimulus is carried directly into the motor areas; without the intervention of ideas or inhibitions, which accounts for the frequent occurrence of outbreaks of violence and destructiveness, followed by complete or partial amnesia as to the acts perpetrated.

Diminution or cessation of sensory and intellectual emotional tone gives rise to the condition known as partial or general *apathy.* A general apathy is frequently observed in neurasthenic insanity and in stuporous states, but it is more common in certain cases of melancholia. Such patients will complain, paradoxically as it may seem, of a painful feeling of having lost all feeling. They say that they feel no affection for their children, no hope of getting well, no pleasure in anything, no grief at the loss of friends, that their hearts are turned to stone.

Sometimes ordinary sensory feeling seems absent also, and they say they can feel neither heat nor cold, nor the pain of a cut or injury. One must distinguish between an apparent apathy and a want of attention consequent upon self-centering of the thoughts on strong delusions and hallucinations.

Partial apathy or limited defects of the emotions, as well as of special and ordinary sensation, are frequently encountered in various grades of congenital idiocy and acquired mental weakness. Defects of the higher forms of intellectual sensory tone, the ethical feelings, which we meet with in some of these cases, constitute the so-called moral insanity.

In certain psychoses a general apathy may be so great and the horizon of intellectual processes so narrowed that the condition amounts to a pseudodementia (Magnan), though there is truly no actual defect of intelligence, the mental functions being merely temporarily inhibited or suspended.

A peculiar mutability or *lability* of affects is not an infrequent phenomenon in insanity. Laughing and crying at the same time is not a rarity in persons who are not insane, being the result of the commingling of pleasant and distressing ideas present at the same moment in consciousness. The emotional pendulum swings quickly from one extreme to the other. Such disequilibration is particularly characteristic of hysteria, and is notable in the hysterical psychoses. But irritability and rapid alternation of cheerful and pathetic affects are also encountered in the most various psychoses. The chronic melancholiac with his sad face and automatically repeating his set phrase, "I am going to be killed," may laugh out suddenly at a funny incident and immediately relapse into his habitual mental attitude. In the same manner the paranoiac may forget momentarily his persecutory delusion. In general paresis this swinging from one emotion to the other in the most rapid manner is extremely characteristic. Mutability of affects is indeed most common in combination with conditions of intellectual defect or mental weakness.

DISORDERS OF THE IDEA-ASSOCIATIONS.

An idea-association is a psychological series, beginning with a stimulus and ending with a movement, between which may be one or two or more memory-pictures, some coming into consciousness, others remaining latent, but all associated by the nerve-fibers running between the ganglion-cells of the cortex in which are deposited the sensory impressions. The selection and serial course of ideas in the stream of thought are determined by fixed laws. One of these is the law of similarity-association—*i. e.*, a sensation induces an idea (seeing a flower gives the idea of a flower) and another latent idea is aroused by this (a rose) because the second memory-picture has marked similarity to the first idea—the rose is remembered or recognized. Every recognition contains a judgment, since a new sensation is seen to be like a former sensation.

Another law is that of simultaneity of reception—*i. e.*, memory-pictures are associated when their sensory stimuli have been received at the same time. For example, the sight of a friend recalls the city, the street, the house where one first saw him, and many others in a highly complex series of associations. Not all of these, however, will arise at sight of him. Perhaps it may be one or two, perhaps others; so that another factor arises—viz., the degree of associative relationship. Still another factor is the feeling (the intellectual sensory tone, the affect) combined with each of the memory-pictures. Those memory-pictures will rise soonest into consciousness which are combined with the liveliest emotions, agreeable or disagreeable, pleasant or painful. Ideas with strong affects have a greater chance in the conflict of ideas to rise up from their latency into consciousness. Still another feature of this scheme is that the latent ideas with their numerous associations influence one another reciprocally, some to excite and some to suppress or inhibit. While simpler ideas are arranged in a sort of serial association one after the other, on a higher plane the successive memory-pictures are bound together into judgments and conclusions. Ziehen cites the example of the simple judgment, "The rose is beautiful," in which we have not these ideas discreetly ranged one after the other, but the ideas "rose," "is," and "beautiful" stand in a thorough relation to one another. This form of idea-association is designated as a judgment-association.

The normal stream of ideas, or idea-association, has a definite swiftness which varies in different individuals and in the same individual at different times. In psychopathology we learn that agreeable or pleasant affects hasten and disagreeable or unpleasant affects retard the flow of thoughts.

The pathological disorders of the idea-association are to be classified as follows:

1. Disorders of memory.
2. Disorders of attention.
3. Accelerated flow of ideas.
4. Diminished flow of ideas.
5. Disturbance of the connections between the ideas of the idea-association (incoherence).
6. Falsification of the judgment-associations (delusions and imperative ideas).
7. Defective judgment-associations (weakness of judgment).

Disorders of Memory.—Recollection according to the principle of similarity-association is the calling up (by a sensation) of a memory-picture of earlier, similar, or identical sensations.

Recollection is disordered or destroyed by loss of the necessary memory-pictures, by any general marked retardation of cortical associations, and by dissociation of the idea-association.

Dissociation is equivalent to incoherence, and when a general incoherence exists, disorder of recollection is the rule. The patient then confounds persons and objects, and often loses the ideas of place and time (a condition for which *unorientation* is the best name). The peculiar paramnesia observed in alcoholic psychoses, especially in the delirium accompanying alcoholic neuritis, is a striking example of this loss of orientation. The mistaking or confounding of persons and things depends upon illusions, delusions, incoherence of ideas, lack of distinctness of the requisite memory-pictures, or, finally, upon voluntary caprices of the patient. In alcoholic paranoia and epileptic insanities, and sometimes in other psychoses, we encounter the so-called "hallucinations of memory"—a bad term for the phenomenon experienced sometimes by normal individuals, of having seen this or that thing, or of having been in the same place before, although in fact the object and place are absolutely new.

Disorders of Attention.—Condillac stated that if amid a multitude of sensations there is one which predominates by its intensity, it is thereby transformed into attention. Ribot [1] regards spontaneous attention as always caused by emotional states. The writer believes, with Ziehen, that attention is never voluntary, but always spontaneous; that it is the awakening of one idea from the impressions of the innumerable sensations impinging on our sensory surfaces. Such attention depends upon several factors. One is intensity. Another is correspondence of the received sensation with some latent memory-picture. A third factor is the affective quality or sensory tone of the sensation. A fourth factor is the combination of latent ideas.

The disorders of attention are morbid diminution and morbid increase. The former is extreme in idiots, and noteworthy in patients dominated by strong hallucinations or overpowering delusions. By pathological increase of attention is meant the crowding of numerous sensations and ideas into consciousness, such as is observed, for instance, in maniacal states.

Accelerated Flow of Ideas.—In the highest degree of pathological increase in the stream of thought we observe not only a rapid concatenation of the associated ideas, but their swift transfer to the cortical motor areas, so that gesticulation, logorrhea, and motor agitation become strikingly prominent. It is an ideomotor excitement. It may be so severe as to present a secondary incoherence. In moderate degrees of acceleration the words spoken by the patient may, by their sound, arouse associations, so that we observe in the speech of the patient a tendency to rhyming assonances and verbigeration. The almost constant combination of augmented flow of thought with an exalted and cheerful mood is interesting and, at the same time, difficult to explain. Some believe that the exaltation is due to the patient's feeling of great facility and fecundity of thought. Others, again, consider the exaltation as the primary phenomenon, and that, as in normal individuals, the exaltation induces the free play of ideas. But it is probable that the cheerful mood and accelerated flight of ideas are simultaneous manifestations of the morbid process.

Diminished Flow of Ideas.—In this symptom we have features quite opposite to those manifested in ideomotor excitement. In the place of increased we have diminished attention to the sensory stimulus, and retarded transfer of the awakened idea-associations to the motor areas (motor inhibition). In any noteworthy inhibition of the flow of thought we observe also difficult and retarded recollection and more or less complete cessation of all voluntary movement. Speech becomes slow, the patient seeking laboriously for words, and these are simply whispered, not spoken aloud. In severe degrees only slight movements of the lips are made, or complete mutism is presented. Sometimes a word or phrase will be repeated monotonously over and over; a single motion of the arm or body may be reiterated for hours (stereotyped movements). The general musculature of the body may be completely relaxed and flaccid (motor-inhibition with resolution) or in a state of tension (catatonic inhibition), or in the condition known as *flexibilitas cerea.* The condition designated as stupor comprises three cardinal symptoms—viz., diminished attention, thought-inhibition, and motor-inhibition. Stupor may be primary or secondary. When secondary, it is ordinarily induced by hallucinations of ecstatic, dreadful, or imperative nature. Stupor from ecstatic hallucinations is frequent in hysteria and epilepsy, and from dreadful hallucinations in melancholia (catatonic syndrome). Primary stupor is another name for primary dementia.

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[1] "The Psychology of Attention."

Depression with thought-inhibition is common, and among the depressed affects associated with it we observe most frequently anxiety. According to the motor symptoms prominent in such cases, such as flaccidity (or resolution), catatonic rigidity, and restlessness, we distinguish three types—viz., melancholia passiva, melancholia attonita, and melancholia agitata. The usual motor inhibition is concealed in melancholia agitata by the expression movements of anguish, such as wringing the hands; picking the fingers, face, or scalp; restless moving to and fro, anteroposterior or lateral oscillations of the body, and the like.

In the diagnosis of thought-inhibition we must be careful to distinguish, in the first place, actual defects of intelligence or conditions of dementia. Then we must distinguish the primary form without affects and with affects, and the form secondary to hallucinations and delusions. Some of the diagnostic criteria are:

Dementia and idiocy are stationary or progressive conditions, while, on the other hand, in thought-inhibition there are transitory variations—intervals of diminished inhibition.

Thought-inhibition is almost always combined with motor-inhibition, while this latter symptom is not observed in defects of intelligence.

The judgment-associations in defective intelligence are also defective, and wrong answers are often given to questions. This is not true of states of thought-inhibition, where correct answers are generally made, if made at all.

Incoherence.—Incoherence is a dissociation of serially related ideas. Such dissociation may involve also the sensations which arouse a series of ideas and the motor sequence of a series of ideas. In a complete general incoherence, then, the patient recognizes neither persons nor objects, calls everything by its wrong name (pseudoparaphasia), uses everything wrongly (pseudo-apraxia), answers questions with absolute irrelevancy, and shows even incoördination and pseudo-ataxia in his movements. When the incoherence is marked in the sensory perceptions, we speak of lack of orientation; it was formerly termed a disorder of self-consciousness. When the motor incoördination is extreme, it may amount to veritable jactitation and pseudochorea. Incoherence is most remarkable, however, in the speech, writing, and mimetic expression of the patient. The gestures and facial movements have no relation to the contents of consciousness; laughter may accompany dreadful hallucinations and a tearful countenance some jocose idea. As regards speech, if the incoherence is of mild degree, only the sentences are misplaced; if of severe degree, the very words in the sentence are jumbled together, and we observe the phenomenon of verbigeration and the manufacture of new words. The handwriting of the patient may present the same incoherence as the speech. The term confusional insanity has been used to describe the form in which the symptoms are want of orientation, incoherence of ideas, and motor incoherence. Incoherence may be primary or secondary, generally the latter. As a primary phenomenon, it is the cardinal symptom of the incoherent form of paranoia. Secondary incoherence is due to extreme rapidity of the stream of ideas, to accumulation of rapidly changing delusions and hallucinations, to strong depressing affects, and finally to actual defect of intelligence. It is often difficult to distinguish primary from secondary incoherence, and far from easy to differentiate the causes of the latter.

Delusions and Imperative Ideas.—Ideas are associated with judgments as to similarity, simultaneity, properties of objects, etc., and such judgment may be correct or erroneous in normal individuals, according to the weakness or strength of judgment, and according to the degree of correspondence between the sensory perceptions and the objects or events of the external world. The normal mind, however, generally corrects its errors of judgment by repeated experience and better education—a physiological process. The pathological errors of judgment are the delusions of the insane. These delusions are usually judgments founded upon incorrect sensory impressions, such as illusions and hallucinations. They are rarely corrected by experience, as is the case with physiological error. But there are many cases in which a definite boundary-line can not be drawn between the delusions of the sane and those of the insane, as, for instance, in the delusions of the superstitious and of spiritualists.

The delusion is the most frequent form of pathological error of judgment, but the imperative idea is also a pathological error of judgment, though less commonly met with. Delusions are seldom influenced by, or, in fact, associated with, attempts at correction by the judgment; whereas imperative ideas are usually recognized as morbid by the patient, but force themselves into consciousness despite the efforts of the judgment to dislodge them.

A delusion may arise in the mind as a primary idea without an incorrect sensory basis, in the same way as an imperative idea. It may be a logical deduction from other delusions, or, as already stated, be the product of illusions or hallucinations. It may be the result of a dream carried over by weakened judgment into the waking life. It may develop, as in melancholia or mania, from the attempts of a patient to explain the origin of his depression or exaltation. Thus, the melancholiac believes that his suffering must be due to his bad conscience, to some sin that he has committed, to some serious disease of his viscera, and the like. The patient with exaltation of his emotional life develops expansive ideas as to his strength, beauty, intellect, wealth, position, and so on. The character of delusions developed in the insane is as multiform as are the ideas in the mind of man.

Depressive delusions are almost always connected with the idea of having committed a sin, of having some disease (hypochondriasis), of having lost all property, or of persecution. Contrasted or antagonistic delusions of grandeur are sometimes observed at the same time in connection with depressive delusions. Thus, one patient, while weeping and wringing her hands, told me she was the queen of the world, but was unable to do her duty because she did not know all languages. Ziehen tells of a patient who said, "I was the Holy Ghost. Had I used my omnipotence, we would all be happy now. But I am cursed. I have killed the Holy Ghost. The whole world is in misery and dread through me." Hypochondriacal delusions generally arise from disorders of common or organic sensibility, cenesthetic sensory impressions, though they also develop from attempts at explanation of a depressed mood and from hallucinations. The patient is certain he has cancer, consumption, syphilis, brain-softening; that he is impotent; that his alimentary canal is closed up; that his brain has been removed; that his viscera and tissues have been metamorphosed into stone, glass, wood, and the like. A peculiar form of hypochondriacal delusion is the so-called micromania not infrequently observed in depressed periods of general paresis. Patients with micromania assert that whole viscera have been removed from their bodies, that their blood is all gone, and that they have been reduced in size. Thus, one patient told me she was so small she could be put into a pill-box. Another said his intestines were absolutely closed up and he should have to be cut open to have the obstacles removed. The delusion of pregnancy arises from perversion of abdominal sensory impressions.

The delusion of persecution differs from the other depressive delusions in that it has to do with the enmity of other persons in the environment, whereas these are concerned altogether with the ego of the patient, his own conscience, his own mind, his own body. The delusion of persecution is important to the general practitioner, because it is very common, because it is met with so often outside of institutions, because it not infrequently leads to assaults and murder, and because its significance in prognosis varies with the species of mental disorder in which it is encountered. It is observed, for instance, in toxic insanities which are curable; in melancholia, in which cure is difficult; and in paranoia, which is incurable. The most common origin of the delusion of persecution is from hallucinations. The patient hears mocking or threatening voices, he tastes poisons in his food, he sees lowering looks and menacing gestures, he feels singular sensations in his body which must be due to irritant poisons thrown upon him or to electricity, or he smells noxious gases. The delusion of persecution may grow out of a series of hypochondriacal delusions, in the attempt of the patient to explain the origin of his miseries. It may arise also from the delusion of having committed a sin or crime, the patient imagining that every one hates him and follows him to punish him. Sometimes these persecutory delusions are referred to the influence of unseen agencies—hypnotism, telepathy, electricity, magnetism. Sometimes they have to do with the property or social position of the patient; he believes his belongings are being stolen, or his character maligned. Sometimes erotic ideas are bound up with persecutory ideas; a woman believes herself secretly cohabited with at night, or even by day, through occult means; a man thinks he is made impotent, that his seminal fluid is being drawn off. Obviously, these latter ideas often rest upon perverted sensory impressions received from the sexual organs. In seeking to discover the origin of the persecution, the patient often at first settles upon some one definite individual, but later, when he finds the methods of persecution innumerable and that his enemies follow him wherever he goes, he can not believe that any one person could do so much; he reaches the conclusion that it must be a wide-spread conspiracy, such as could be carried out only by some large affiliation of persons, such as societies of Freemasons, anarchists, Jesuits, lawyers, and police. The delusion of persecution occasionally develops from a delusion of grandeur; the patient believes he is persecuted because of his wealth or exalted position. More often, however, the contrary is the case, the patient coming to believe himself some extraordinary personage because of the persecutions to which he is subjected.

Another interesting form of depressed delusion is that of negation (*délire de négation géneralisé*), which has its origin usually from an idea of having sinned. The patient thinks he must be the devil himself, his sin is so great; consequently he can never die, he must suffer forever; then, with the growing idea of the enormity of his sin, he comes to believe that God and mankind and the world exist no more.

Delusions of grandeur vary from simple, expansive ideas of the patient's importance, prerogatives, and powers, to delusions of being inventors, geniuses, prophets, reformers, titled and royal personages, and even Christ, God, and the mother of God. Besides his own personality, his environment may be vested with grandiose qualities—his room a palace, his straw hat a crown, pebbles diamonds, his children princesses, and so on. A peculiarity of the ideas of grandeur observed in general paresis, which is quite pathognomonic, is their enormity or, rather, monstrosity. It is not enough to be wealthy, but sextillions of planets can not hold the gold and jewels. It is not sufficient to have a dozen children, but billions of children are given birth to nightly by his innumerable wives. He will make a new Niagara, by bringing the Pacific Ocean over the Andes. Should sexual ideas prevail, he may say that his penis is a mile long, and his testicles are huge diamonds. He will move the asylum across the United States on a road of solid gold. Such enormities betoken great weakening of the intellect and judgment.

Primary delusions conduce more to fixity than delusions secondary to hallucinations. The latter, depending as they do upon the stability or instability of the morbid sensory impressions, change with these. When delusions become fixed, they tend to crystallize or become systematized. Systematization consists of combining with the fixed idea complementary delusions in a more or less logical order or of the fantastic elaboration of the original delusion. The degree of organization and perfection of the delusional structure will depend upon fancy, logical faculty, social position, and education of the patient. The most common form of systematization is in the development of secondary grandiose ideas upon a persecutory basis. But almost any of the depressed and exalted delusions previously described may become fixed, systematized, and permanent through the life of the patient.

Delusions may have a retroactive effect in awakening sensory impressions, instead of being aroused by them—that is, may induce illusions and hallucinations. For example, the persecuted patient perceives voices, odors, tastes, pains, etc., often because of his mind being in a state of expectant attention.

Imperative ideas force themselves into consciousness in spite of the efforts of the patient—who recognizes their morbid character—to correct them. They are accompanied, almost without exception, by a depressive affect, a painful sensory tone. They are extremely common in neurasthenia. Senseless phrases or doggerel repeat themselves over and over in the patient's mind. The many varieties of phobia are familiar examples of imperative ideas in neurasthenics (agoraphobia, claustrophobia, mysophobia, etc.). Imperative ideas are also observed in melancholia and in a form of insanity which has been designated as insanity from imperative ideas. In very rare instances they are encountered in early stages of general paresis. They always develop on the basis of a congenital or acquired neuropathic or psychopathic constitution, and are apt to become obstinate features in the mental organization. Almost every imperative idea has its inception in some sort of sensory impression, and the idea may lead to compulsory actions on the part of the patient. But between the imperative idea and the consequent action there is generally a play of judgment, a faltering between the imperative idea and antagonistic or inhibiting concepts. For instance, the patient feels a compulsion to lock a door which he feels sure he has already locked. After an inward debate as to whether he should go back and assure himself that it is locked, which may last many minutes or longer, he goes to lock it, and on leaving the door again the imperative idea arises that it is not locked. The same play of antithetic ideas may occur in reference to anything—the addressing of a letter, the return of a book to a shelf, acts of dressing and undressing, the crossing of a street, etc. In some cases the imperative idea takes the form of compulsion to jump from a height, to laugh in unseemly places; or obscene and sacrilegious words, sentences, and fancies may thrust themselves obstinately into the consciousness. For example, a gentleman, and a good Christian, came to me recently overwhelmed with the sacrilegious conceptions which first came to him at a church-service a week or two before—ideas of cohabitation with the Virgin Mary and filthy expressions in relation to Christ. A lady consulted me about a morbid fear that she had of canary birds. She could not enter a house or hotel in which there was a canary bird, because she was afraid that bird-seed might get about and in some way get into her mouth, be swallowed, and grow in her stomach. The contents of these imperative concepts are as varied as those of delusions, though they are almost, without exception, trivial or unpleasant.

Folie du doute is a form of mental disorder in which compulsory ideas assert themselves in the form of questions, religious, metaphysical, or in regard to the most trivial things or events (Shall I do this or that? Why is the table round? Why is the chair by the bed? Why are two and two four?). One young lady is so incapable of deciding any question that comes up in her mind that she does not know whether she ought to dress or undress, go to bed, eat, sleep, pray, or consult a doctor. Every trivial question of the day requires hours of painful and agonizing debate in her mind.

Imperative ideas frequently impel to compulsory speech and actions. Coprolalia is a not uncommon form of imperative speech in which the patient is impelled to the utterance of obscene words. Quite analogously the patient may be made to make grimaces, or may develop the so-called *maladie des tics*.

Weakness of Judgment.—Innumerable memory-pictures and associated ideas take part in the process of comparison and decision which we know as judgment. Hence any disorder of memory and of its associations, such as loss, defect, or perversions (delusions, hallucinations, or illusions), must naturally influence the character of the judgment. One of the common conditions which impairs judgment is, therefore, intellectual defect, such as congenital or acquired mental weakness. The criteria of idiocy and dementia are poverty of ideas and idea-associations and weakness of judgment. When delusions or imperative ideas exist, the errors of judgment are due to the overriding and eclipsing by single ideas and idea-associations of all others which would in the normal mind give balance, control, and revision to the judgment. Defective judgment varies in degree from a slight loss of the critical faculty to complete deficiency. When the judgment is markedly defective, it depends upon actual organic changes in the brain, such as we observe in idiocy, terminal dementia, senile dementia, and general paresis, and hence as a symptom it is far more ominous than delusions and imperative ideas, which usually rest upon a functional pathological basis. Its significance, then, demands a careful differentiation of this symptom from others with which it might be confused, such as incoherence and thought-inhibition. In incoherence the threads of thought are constantly lost. In thought-inhibition there are a depressive affect and extraordinary slowness of association with correspondingly tardy answers, and, besides, there are variations of depth of inhibition, so that at times complicated answers and judgments are readily given. In actual weakness of judgment the judgments rendered are false, and the more incorrect, the more complicated the questions.

DISORDERS OF ACTIONS.

The actions or conduct of a patient depend directly and necessarily upon pathological elements in some part of the psychological processes —sensation, memory-pictures, idea-associations, and their emotional affects. They may be classified, following Ziehen, as—

1. Actions induced by sensory disorders.
2. Actions induced by disorders of memory.
3. Actions induced by disorders of the emotions.
4. Actions induced by disorders of the idea-association.

Actions Induced by Sensory Disorders.—Hallucinations and illusions affect the conduct of a patient often markedly, and their influence is always greater than that of normal sensations. Their dominance is the greater in proportion to their number and to the rapidity of their accumulation. Hallucinations gathered slowly in the course of weeks or months, while they may not be corrected, are at least subject to a certain amount of control by the inhibition of normal ideas. In the most chronic forms of hallucination the voices, common sensations, and visions tend to be ignored and to influence to a very slight degree the conduct of the patient. A very important practical feature in regard to hallucinations and their effects upon conduct is their uncertainty. They are never to be reckoned with, and one can never know what sudden violence or destructiveness may result from new hallucinations rising in the patient's brain.

Actions Induced by Defects of Memory.—These are observed in congenital or acquired weak-mindedness, where the conduct is directly ordered by sensory impressions, without that intervention of the play of motives which we observe in normal individuals. They are more like the actions of the lower animals, which may be complete enough in their way, but are not motived by complicated abstract conceptions, because these are wanting.

Actions Induced by Disorders of the Emotions.—As already elsewhere intimated, simple depressed emotions are accompanied by a general motor inhibition, and simple exalted emotions by a general motor agitation. But when the depressed affect attains to the degree of anxious dread, we may have a restlessness, a desire for flight, which in itself amounts to a motor agitation. This anxious state often leads to suicidal attempts, and even to homicidal assaults, arson, and other forms of crime and violence. The whole nervous system seems to be in such a state of tension that only an explosion can give relief.

In apathetic conditions action is reduced to its minimum.

Where the higher affects, which are at the basis of ethical concepts, are absent or lost, as in congenital or acquired states of mental weakness, crimes against person and property are common.

In conditions of anger and rage there is at first a brief period of speechlessness and immobility, followed by an explosion of blind and violent motor excitement, in which the most dangerous assaults may be made.

In conditions of changeability or lability of the emotions, we observe analogous motor states—sudden changes from weeping and wailing to boisterous cheerfulness, and vice versâ.

A study of emotional expression is of particular diagnostic value in insanity, but the features of such expression and gesticulation are so well known that they need no detailed description here. Each mood, be it simple depression, anxious terror, excitation, anger, apathy, or emotional lability, has its own familiar motor habiliments.

Actions Induced by Disorders of the Idea-association or Stream of Thought.—Under this heading are gathered the multiform modes of action caused by increase in the flow of ideas, retardation of the stream of thought, incoherence, delusions, imperative ideas, and weakness of judgment.

In increased rapidity of the flow of ideas we note motor agitation or morbid impulse to movement, varying from simple talkativeness, with active play of expression, to loud garrulity, grimaces, gesticulation, busy walking about, running, dancing, and, in extreme degrees, to undressing, destructiveness of clothing, bedding, furniture, and blind throwing about of the body in every conceivable way. This so-called primary motor agitation should be distinguished from the motor agitation which is secondary to crowding hallucinations (hallucinatory agitation) and to emotions like terror and anger (affective agitation).

The behavior of the movements in regard to retarded flow of thought has already been briefly alluded to. There is a general motor inhibition, varying from simple slowness and difficulty of executing any movement, whether of speech or other muscles, to a complete cessation of voluntary movements, a stuporous or attonitous condition, in which the muscles may be absolutely at rest and flaccid or, on the other hand, in a condition of catatonic tension. In true catatonic tension every attempt at passive movement is resisted, but in another form of this there is a waxy flexibility of the muscles, so that the limbs yield readily to any passive motion, remaining in whatever position the physician desires to place them. Occasionally one encounters in cases of retarded idea-associations, as an expression of motor inhibition, a tendency to the repetition of some restricted voluntary movement in a rhythmical, stereotyped way for days, weeks, months at a time. Such stereotyped motions may be simple anteroposterior oscillations, lateral oscillations, whirling, walking to and fro or in a circle, waving the hands rhythmically—forms of tics exceedingly common in idiocy and imbecility, but common enough in melancholias and terminal dementias. The repetition of stereotyped or automatic phrases is analogous in character to such morbid movements. Motor inhibition is primary or secondary. The primary form is generally a simple resolution or flaccidity, occasionally a slight catatonic tension or *flexibilitas cerea*. Secondary motor inhibition is due to hallucinations, delusions, and states of mental weakness.

Incoherence of ideas leads to a dissociation also in the motor expressions of ideas, parapraxia, paramimia, incoördination, pseudo-ataxia, incoherent agitation, chorea magna, and jactitation. Such motor agitation may be primary or may be the secondary result of innumerable clashing hallucinations and delusions, rapidity of the flight of ideas or of intellectual defects.

Grandiose delusions exert their own peculiar influence on the demeanor and speech of the patient, according to the contents of the exalted ideas. We observe the proud bearing; the self-sufficient, haughty, or secret smile; the withdrawing from others; the tendency to decoration of the person; the attempts to act the parts of the personage he imagines himself to be; the striking peculiarities of handwriting. In some instances delusions of grandeur lead to homicidal, rarely suicidal, attempts (self-crucifixion with the delusion of being Christ). Grandiose erotic ideas sometimes occasion masturbation. Coprophagy and other filthy habits may depend upon grandiose delusions as to extraordinary virtues of the patient's excretions.

In depressed delusions, particularly as regards ideas of sin and poverty, we observe the characteristic melancholy facial expression and attitudes. Attempts at suicide are frequent, and sometimes self-mutilation. Abstention from food is especially common with the delusion of poverty, the patient feeling that he can not pay for anything.

Hypochondriacal ideas influence markedly the patient's actions and conduct. The hypochondriac may neglect every duty in the constant contemplation of his symptoms. He reads medical books, goes from one physician to another, takes to his bed perhaps permanently, and so on. The effects of hypochondriasis on motor functions are frequently remarkable, leading sometimes to astasia or abasia, or both; to hypochondriacal ataxia, tremor, or convulsive movements of the extremities. These hypochondriacal motor conditions are always the result of a series of morbid judgments on a hypochondriacal basis, and are to be distinguished from similar hysterical states which have an autochthonous origin without any antecedent conscious reasoning process.

The persecutory delusions lead to systems of self-protection of the most varied kind. Barricades, stopping up of cracks and keyholes, the wearing of peculiar clothing (silk, paper, etc., for instance, as a guard against electrical shocks), avoiding of food and drink which are suspected of containing poison, arming with weapons, frequent change of servants or residence, and complaints to the police or judicial authorities. Homicide is common in these cases.

Imperative ideas lead to imperative movements and actions, and generally in spite of the well-preserved consciousness and judgment of the patient. Such imperative actions are as various in character as the imperative ideas to which they correspond.[1]

Accompanying Physical Disorders in Insanity.—Among the many somatic symptoms which may complicate or accompany psychoses are chiefly to be mentioned the following:

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[1] The foregoing account of the psychopathology of insanity is largely a presentation of the views of Ziehen, to whose excellent work the author must refer readers for greater detail.

1. Motor disorders.
2. Sensory disorders.
3. Reflex disorders.
4. Trophic disorders.
5. Secretory and excretory disorders.
6. Temperature disorders.
7. Vascular disorders.

Motor Disorders.—These may be manifested in the form of morbid movements or paralysis. In the first category are assembled such symptoms as epilepsy, convulsions, chorea, choreiform movements, tremor, tics, ataxia, masticatory spasm, and the like. The following table, modified from Ziehen, gives a general summary of the paralytic symptoms noted in insanity:

FORM OF PARALYSIS.	CHARACTER.	TROPHIC DISTURBANCES.	SPASTICITY OR FLACCIDITY.	SENSORY DISORDERS.	DEEP REFLEXES.
Hypochondriacal.	Usually limited to a certain form of movement.	No atrophy.	Flaccidity.	None.	Normal.
Hysterical.	Monoplegia, hemiplegia, or paraplegia.	Disuse atrophy.	Frequently contractures.	Hemianesthesias, etc.	Normal or hypertypical.
Cortical.	Monoplegia or hemiplegia.	Disuse atrophy.	Rigidity, contractures, local spasms.	Paresthesias, occasionally anesthesias.	Exaggerated usually.
Pyramidal tract.	Hemiplegia or paraplegia.	Disuse atrophy.	Spasticity, contractures frequently.	Occasionally anesthesia, hemianopsia, etc.	Exaggerated.
Peripheral.	Multiple or single.	True atrophy with degenerative reaction.	Flaccidity.	Hyperesthesias, stocking and glove areas of anesthesias often.	Lost.

Sensory Disorders.—Anesthesias and hyperesthesias have already been mentioned, but hyperalgesias and paresthesias of divers kinds are encountered among the psychoses, such as headache, migraine, neuralgias, feeling of fullness in the head, scotomata, tinnitus aurium, and so on. Neuralgia is occasionally a cause of insanity. Migraine is a frequent precursor of general paresis and concomitant of epilepsy. Lightning pains are noted in tabic types of dementia paralytica. Neurasthenic pains and paresthesias in the extremities, spine, and head are found in neurasthenic forms of insanity. Where hysteria complicates a psychosis, there are often observed the sensory disturbances characteristic of that malady.

Reflex Disorders.—Changes in the reflexes are important in but a few forms of insanity. In paralytic dementia we observe nearly always exaggerated tendon-reflexes, but in tabic types they are lost. They are lost also in psychoses complicated with multiple neuritis, and frequently in cases with diabetes, and in morphinomania. The deep reflexes are exaggerated in senile dementia, many acute affective insanities, hysteria, epilepsy, and in patients with accompanying multiple sclerosis. The state of the superficial reflexes possesses little significance, except in insanity associated with hysteria and organic disorders of the brain, spinal cord, or peripheral nerves.

The Argyll-Robertson pupil is met with almost constantly in general paresis. The pupils in all cases of insanity should be examined as to their equality, size, and reaction to light, and in accommodation. Loss of reaction to light may be observed, besides, in general paresis, in syphilitic insanities, senile insanity, and in some alcoholic cases; it means organic disease of the brain. In rare instances a transitory rigidity of the pupil occurs in epilepsy and morphinomania. Inequality of pupils is very common in organic and occasional in functional insanities.

Trophic Disorders.—General disturbances of nutrition, variations in bodily weight, are commonly noted, and possess considerable significance. Thus, rapid increase in weight is characteristic of the progress of an acute psychosis to terminal dementia; if, however, it accompanies an improvement in mental symptoms, it betokens convalescence. In some cases enormous decrease in weight, in association with pernicious anemia, leads to a fatal termination. Certain forms of insanity, especially organic, notably paralytic dementia, present a remarkable trophic disturbance in the bones, a fragilitas ossium, inducing easy fracture. Decubitus is observed in bedridden insane patients, particularly paretics.

Hematoma auris, othematoma, or the "insane ear," is a deformity of the ear produced by a hemorrhage into the substance of the auricle, usually between the perichondrium and the cartilage. It is undoubtedly traumatic in its origin, but there is fundamentally some change in the vascular walls in certain cases of chronic insanity, rendering them fragile and easily ruptured by the most trivial pressure or injury. Such effusions of blood do occur in normal individuals (athletes and boxers), but always from severe trauma. The frequency of hematoma auris in general paralysis, and in many chronic forms of insanity is only explicable on the hypothesis of some trophic change in the vessel-walls.

Secretory Disorders.—The secretion of tears is generally reduced or absent in melancholia.

The saliva may be diminished in quantity in melancholia. More often in many forms of insanity it is increased, the excessive secretion amounting sometimes to a sialorrhea. The increase is due to constant mastication, to illusions and hallucinations of taste, and sometimes to irritative stimuli in the secretory centers. Drooling may give the appearance of an increase of salivary flow, because of relaxation of the oral and buccal muscles, or because the secretion is not swallowed.

Diminution or increase of hydrochloric acid in the gastric juice is noted in many cases of insanity, and the quantity may be determined by the Sjöqvist method. Hypochlorhydria exists in common in states of congenital and acquired intellectual defect and in general paresis. Hyperchlorhydria is not infrequently met with in cardialgic attacks, after epileptic seizures, and in catatonic conditions.

As regards the *urine*, quantitative and qualitative changes are very common in insanity. These changes may be the expression of abnormal metabolism in the central nervous system, of abnormal metabolism in other parts of the body induced by disease of the central nervous system, or of vasomotor changes in the kidneys brought about by the psychoneurosis. Polyuria is observed in many organic psychoses and in hysterical complications. Oliguria is characteristic of melancholy and stuporous conditions. In hysterical insanity there is frequently an alternation between oliguria and polyuria.

As regards the qualitative changes in the urine of the insane, we are year by year recognizing more and more the importance of investigation in this direction. There is no doubt that the deeper our researches go into the chemistry of metabolism and catabolism, the nearer do we attain to a better understanding of the mysterious nutritional processes that have to do with the construction of the blood and that underlie so many psychoses. Albumin, peptone, and propeptone are found not infrequently in the urine of cases of organic insanity, in delirium tremens, in epilepsy, and in acute mania. Their presence is often transitory, and unaccompanied by renal disease. Hyalin cylinders are also often observed in severely excited conditions.

Excessive phosphaturia is noteworthy in many cases of great cerebral excitement, and after epileptiform and apoplectiform seizures. In chronic brain disorders the quantity of phosphoric acid is diminished below the normal.

The chlorids are lessened in quantity in melancholia. They are increased in the early stages of paresis, but diminish with the progress of the disease to dementia.

Sulphates and the aromatic ethereal sulphates (the latter being the product of destructive proteid metabolism) are increased in febrile conditions, and in conditions attended with much tissue-waste.

Urea is also representative of destructive proteid metabolism, and is an index of the general nitrogenous metabolism of the body. It is increased in conditions associated with tissue-waste, diminished in states of malnutrition. Uric acid and the urates have much the same relation.

Oxaluria (any increase above the normal amount excreted in twenty-four hours—viz., $\frac{3}{10}$ of a grain) is observed in certain nervous and mental disorders, but its precise significance still requires determination.

Urobilinuria and bilirubinuria have occasionally been noted in general paresis.

Glycosuria, with or without polyuria, has often been observed in various organic psychoses. It may be intermittent, transitory, or permanent.

Acetonuria is encountered in general paresis and epilepsy at times, as also in psychoses attended with malnutrition, as, for instance, melancholia.

Indican should be sought for, as it is an indication of albuminous putrefaction. It is significant of auto-intoxication.

There is a wide region open to the pathological chemist for discoveries in the feces, as well as the urine, of relations between metabolism and psycopathic disorders.

Menstruation is often disordered in insanity. Amenorrhea is the rule in acute psychoses of any form, due undoubtedly to profound changes in the general nervous system influencing the spinal centers for ovulation and menstruation. The cessation of menstruation with the onset of an acute psychosis is often mistakenly supposed by the laity to show some etiological relation between the genital organs and the insanity. The return of the menses is one of the early signs of convalescence from acute mania and acute melancholia. Naturally, it would not be correct to ascribe amenorrhea in all cases to simply nervous inhibition, because it may arise in all kinds of psychoses as the result of actual genital disease or of marked anemia.

Temperature-changes in Insanity.—The physiological oscillations of temperature are greater and more irregular in the insane than in normal individuals. In general, however, insanity may be said to run a non-febrile course.

Subnormal temperatures are frequently observed in melancholia, stuporous states, general paresis, idiocy, and occasionally in conditions of great excitement. In these last they are apt to indicate approaching collapse.

Hypernormal temperatures are found in many psychoses, sometimes from very slight peripheral irritations, such as retention of urine, gastric catarrh, constipation, mild bronchitis, decubitus, sometimes from organic changes in thermogenic centers. Hysterical complications may be associated with hysterical fever. Motor agitation in mania, acute paranoia, melancholia, and so on, may, if marked, give rise to febrile symptoms. The status epilepticus and convulsive seizures of general paresis increase the temperature, as a rule, to a noteworthy degree. Many writers have described diurnal oscillations of temperature, variations from day to day, asymmetrical axillary temperature, and general subnormal and hypernormal conditions of temperature in paralytic dementia; and some years ago, in association with Dr. Langdon, I undertook a verification of these statements at the Hudson River State Hospital for the Insane.[1] These are the conclusions we drew from a study of the temperature in twenty-five cases of general paresis:

1. As regards the average bodily temperature, we find it to correspond to physiological norms. The statements of our predecessors as to hyperpyrexic or subnormal averages can not be sustained.

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[1] "A Study of the Temperature in Twenty-five Cases of General Paralysis of the Insane," "Journal of Nervous and Mental Diseases," Nov., 1893.

2. The diurnal oscillations of temperature in paretics also correspond to physiological norms. The statements to be found in literature as to extraordinary daily variations being frequent in these cases are absolutely erroneous.

3. Asymmetrical axillary differences are so small that they can not be considered as abnormal, and certainly not of any diagnostic significance.

4. When unusual variations of temperature occur in general paretics, their cause must be sought for in conditions not related to the pathological phenomena of paralytic dementia, but depending upon thermogenic features unrecognized by the physician, or "masked" by the mental state of the patient. Thus, in case two of our series, an increasing hyperpyrexia was noted during the second week's observations, but the pneumonia causing it was "masked" until the fifth or sixth day, the patient dying on the sixth day. Again, in case ten, where the highest single daily oscillation was 3.4 degrees, and the average daily oscillation for the week 2.2 degrees, the patient suffered from bed-sores, which undoubtedly produced some septicemia. That variations of temperature take place in connection with the paralytic and convulsive seizures of these cases we do not gainsay.

Vascular Disorders.—The action of the heart and vessels is often influenced by insanity. The pulse is subject to acceleration in excited and neurasthenic states, and to retardation in stuporous conditions. Variations in arterial tension are particularly noticeable at times; arterial spasm in any psychosis, but especially in melancholia, depressed types of general paresis, and in paranoia; arterial paralysis as a sequel to this. No doubt strong mental shocks and depressive or exalting affects are associated with anomalies of the vasomotor innervation. Perhaps many psychoses depend upon cerebral angioneuroses. The apoplectiform, epileptiform, and maniacal seizures of general paresis are believed to have their origin in these. Precordial anxiety, the neuropathic cervical globus, and other paresthetic and paralgesic sensations in the domain of the vagus, are also, in all likelihood, due to angioneurotic conditions.

CHAPTER IV.

EXAMINATION OF THE PATIENT; DIAGNOSIS; COURSE OF THE DISEASE; PROGNOSIS.

In most cases of insanity the diagnosis of the presence of a psychosis and also of the form is by no means difficult; but there are many in which this is not the case. Medicolegal cases especially require most careful and painstaking investigation in order to arrive at exact knowledge of their mental state. It is well to follow at all times some fixed scheme of examination, and the writer advises the following:

1. Hereditary factors.
2. Pregnancy and parturition of mother.
3. Convulsions or other nervous disorders in infancy.
4. At what age did patient walk, speak, and complete dentition?
5. Degree and character of education.
6. Rachitis or febrile disease in childhood.
7. Character and temperament in childhood.
8. Period of puberty—Was its development normal?
9. Strength of sexual instinct at this period? Masturbation?
10. Occupation during adolescence.
11. Character, temperament, religion, physical condition, diseases during adolescence.
12. Sexual relations—excessive, illicit; marriage—venereal disease, puerperium.
13. Intemperance in the use of alcohol or drugs, overwork, shock, trauma to the head, infectious diseases, genital disorders.
14. Psycopathic constitution, previous attacks.
15. History of present attack.
16. Probable etiology.

Physical Condition.—1. Height, weight, nutrition, circulation, pulse, temperature.

2. Cranial measurements. } See chapter on Etiology.
3. Stigmata of degeneration. }
4. Condition of heart, lungs, alimentary canal, genito-urinary organs.
5. Reflexes, pupillary and tendon.
6. Condition of cranial nerves.
7. Motor symptoms—paralysis or morbid movement, gait.
8. Common sensory symptoms—paresthesia, hyperesthesia, anesthesia.
9. Special sensory symptoms—sight, hearing, smell, taste, field of vision.
10. Speech disorders—stammering, syllabic articulation, paretic speech, motor or sensory aphasia, agraphia, apraxia, verbigeration.
11. Sleep.
12. Expression, gesticulation, attitude.

Psychic Condition.—1. Mode of speech—accelerated, retarded, mute, incoherent.

2. Conduct—Does patient care for his person and dress? Does he attend to his ordinary duties? Is he excited and active, or depressed and quiet?
3. Illusions or hallucinations?
4. Mood—Is the fundamental mood depressed, exalted, irritable, changeable, apathetic?
5. Ethical feelings—What is the state of his moral conceptions and judgments?
6. Attention—Does the patient note what goes on about him, etc.?

7. Memory—Weakened or increased as regards long past and recent events. Test mathematical, geographical, and historical knowledge. Test dates, ages and names of members of family, the events of the past few days, etc.

8. Idea-association—Is there apraxia or parapraxia? Does the patient orient himself as to place, time, and objects and persons about him? Is the flow of ideas accelerated, retarded, or incoherent?

9. Judgment—How does he explain his own morbid condition and his relation to his surroundings? What does he busy himself with now, and what are his plans and purposes for the future?

10. Delusions and imperative ideas.

From the scheme just given, it will be seen that the study of a case of insanity, and the taking of the history of the antecedent factors and of the psychic disorder itself, involve much more elaborate attention than is the case with the ordinary physical diseases which one meets with in practice. In medicolegal cases we have to guard against several sources of error in our diagnosis, among which are the concealment of delusions by an actually insane patient and the simulation of insanity by a sane criminal. The forms of insanity usually simulated, because of the facility of so doing, are a maniacal state, dementia or stuporous melancholia, and epilepsy with insanity. Only one with excellent knowledge of the symptoms of insanity can simulate any form of psychic disorder so well as to defy the skill of the physician familiar with mental diseases.

In general practice it sometimes occurs that peculiar forms of delirium incident to severe visceral disease may be at first mistaken for insanity. Thus I have, on a number of occasions, been called upon to assist in the commitment of patients to asylums, where careful examination showed the existence of either a transitory delirium in association with an apoplectiform or other organic lesion of the brain, or a delirium from some such visceral condition as Bright's disease. Delirium of this kind is distinguished, first, by the discovery of the associated and causative organic disease, and, secondly, by the usual non-conformity of the delirium to any special type of psychosis.

Were I to formulate a series of rules to guide the examiner in his investigation of the mental condition of a patient, they would be somewhat as follows; yet it is to be remembered that these are not fixed rules, but subject to much modification by the tact, good judgment, and common sense of the examiner:

1. It is to be presumed that previous to seeing the patient the examiner has fully informed himself of all of the facts to be furnished by relatives or friends, and has, when possible, inspected letters and other writings, which so often prove fruitful sources of information.

2. Go to the patient as a physician, and not under the pretense of being something else—a device so often suggested by the family and friends.

3. Proceed to the physical examination of the patient, during which tactful questioning will determine the direction to follow in further inquiries.

4. Gain the good will of the patient by kindness and consideration.

5. Even if the patient is distrustful and uncommunicative, be politely persistent, and prolong the first examination, even to the extent of trying the patient, until the object is attained; for many patients will, when fatigued, finally yield to the friendly insistence of the examiner.

6. If one examination is insufficient, however, have as many interviews as are requisite for the purpose in view—a careful scientific diagnosis. In medicolegal investigations this is especially necessary.

Course of Insanity.—In all forms of insanity we should seek to divide the symptoms presented into primary and secondary classes, not only because of the interest of so doing, but because of the value of the division in the matter of diagnosis. For instance, in some cases we discover hallucinations and illusions to be the primary symptoms, and, in addition to these, we observe, as secondary symptoms, delusions, diminished attention, inhibition of thought, motor inhibition, and an anxious state; the condition is that of hallucinatory stupor.

Furthermore, we will divide insanities into acute and chronic forms, referring usually rather to the rapid or slow mode of onset than to the duration of the disease. But sometimes these qualifications are employed in connection with the duration of the insanity. Thus, it is customary in some asylums to designate mania or melancholia as acute for one year, at the expiration of which the term chronic is used. Remissions in insanity are periods of improvement or apparent recovery. Intermissions, or lucid intervals, are periods of complete recovery between attacks.

Mental disorders, owing to the delicate nature of the physical structures in which morbid changes take place, are prone to run a longer course than diseases of other than nervous tissues. It is true that transitory insanity and acute delirium may complete their course in a few days, and that acute mania or melancholia may recover in a month. But three to six months is a better average for acute mania and melancholia. Chronic insanities may last indefinitely, for, strangely enough, there seems to be little in these slow alterations of the mind to influence vitality. The average life of chronic lunatics in asylums is said to be some thirteen years, to which, no doubt, the regularity of life in institutions contributes. Many cases of marked chronic insanity last twenty, thirty, even fifty, years, and over.

In any given case of curable insanity, we usually discover, on examination, certain stages of evolution, prodromata, complete development, and involution or convalescence. Régis has devised charts which show diagrammatically the daily range of affects in cases of acute mania, acute melancholia, and circular insanity. In acute mania, for instance, we note a brief prodromal stage of depression, followed by gradually increasing maniacal excitement, then by gradual subsidence of the exalted stage to the normal line. He should have added before the normal termination an aftermath of a peculiar tearful irritability noticeable in these cases. An analogous reactive condition is observed near the termination of acute melancholia in recovery—a certain morbid cheerfulness.

Insanity terminates in recovery, in recovery with defect, in chronic persistence of the same symptoms as at first manifested, in secondary or terminal dementia, or in death. Death often arises from exhaustion due to ideomotor excitement, and in organic psychoses from associated disease of the central nervous system, but, as a rule, death in insanity is not a consequence of the mental disorder, but of intercurrent or incidental disease, such as pneumonia, tuberculosis, etc. There is great variation as regards curability in the different types of psychoses. For instance, ninety per cent. of cases of simple melancholia and seventy per cent. of acute mania recover, while general paresis is invariably fatal.

Prognosis.—The prognosis will depend upon several factors, among which the most important are the type of insanity presented, the course, the character of certain symptoms, and the intellectual development of the patient.

As regards the type of insanity presented, affective insanities (mania and melancholia) are the most curable. Systematized chronic insanities (paranoia) and cyclical forms rarely recover. Organic insanities are nearly always incurable, if not fatal. In mania and melancholia, the acuter the onset and the more rapid and intense the appearance of the symptoms, the better the prognosis. Slow development and progress and partial remissions are unfavorable.

The etiology has considerable influence on prognosis. A transitory cause, like fright or anemia, is favorable. The older the patient, the worse the prognosis. Hereditary taint is not at all unfavorable as regards recovery from an attack of acute insanity, but very much so as regards the probability of relapse. On the other hand, a hereditary taint in insanity of slow inception is of serious significance. Alcohol and morphin, as etiological factors, influence prognosis unfavorably, because of the psychic degeneration they superinduce. Syphilis is not unfavorable if the insanity is due to direct specific disorders of the brain, but of bad import if due to the later, chronic, diffuse, specific alterations in the central nervous system.

Special symptoms, such as defect in the intellectual processes, systematization of delusions, primary delusions, paralysis, and convulsions, are significant of incurability.

The lower the grade of intellectual development in the patient, the greater the danger of termination in dementia.

CHAPTER V.

GENERAL TREATMENT OF INSANITY.

It is not so long a time since the insane in Christendom were believed to be possessed of devils and accursed. On the other hand, in certain parts of heathendom (among the Mohammedans) it was supposed that the souls of the insane had been removed early by God as a special mark of favor, and that they were, therefore, blessed. Medieval treatment was founded upon the curious pathology just described. One portion of the world ducked, whipped, tortured, chained in dungeons, and occasionally burned, the insane. The heathen treated their insane, upon the whole, comparatively well.

After a time, many of the therapeutic measures employed by the Europeans of the middle ages were abandoned as unsatisfactory. But society still had to be protected; so the insane were fettered in the cells of jails and fortresses and solitary towers, until a realizing sense of the inhumanity of such treatment struck a responsive chord somewhere in the breast of a Tuke, a Connolly, a Pinel, a Rush, a Kirkbride, an Earle, and doubtless other, but unknown, immortals both before and after them.

Insanity thus gradually came to be looked upon as a disease, and not a penal offense, and, instead of prisons, special buildings were set apart for the particular custody of the insane. The great object of the asylums at first was to afford protection to society from lunatics, to protect them from themselves, and to provide for their care and support, when at public cost, in an economical manner. A hundred years ago, however, the asylum was still a species of jail, for its evolution had not yet proceeded far. Dungeons and iron chains and staples in stone walls and stone floors were still in use in many places. Indeed, it is scarcely over eighty years since Norris, a patient in Bedlam (Bethlehem Hospital), in the great Christian city of London, was kept for twelve years in a cell, with an iron collar riveted around his neck and iron bands and rings around his wrists, arms, and ankles, the neck being fastened to the wall and the leg to a rude box of filthy straw.

Asylums have, at the present time, come to be recognized as hospitals, and they are approaching nearer to that ideal every year. Occasionally, one finds among them some rudimentary appendage which is reminiscent of the embryonal stage of their evolution; but this is, fortunately, rare. The well-conducted hospital for the insane, to-day, is different from the asylum of years ago; the depressing, barren halls and wards and naked floors have given place to pleasantly furnished and carpeted, cheerful-looking parlors, sitting-rooms, and bed-rooms; muffs and strait-jackets have disappeared; the unintelligent attendant has, in many instances, given place to the trained nurse; every new means of treatment is carried out to the best of the ability of the asylum physicians; schools, employment, theatricals, music, and out-of-door walks are provided in the place of the old, deadly monotony, and, in fact, the asylum has gradually undergone a metamorphosis, until its character has completely changed. There are, to be sure, not many perfectly ideal institutions as yet in existence, but there are some which approach very nearly to it, as, for instance, that at Alt-Scherbitz, near Leipzig, and the new asylum at Rome, both of which I visited and described in 1887.[1] These are, of course, constructed on the cottage and pavilion plan, so arranged as to impress one as small colonies or villages, with separate buildings for those merely there for custody because of dangerous propensities, those brought there to be cared for kindly during the remainder of their useless lives, those who carry on various occupations, and, finally, for such as enter particularly to secure treatment for the brain-malady which has bereft them temporarily of their reason. The colony system of caring for the dependent classes—which the writer thinks should ultimately be adopted for all kinds of defectives—is well exemplified by the Craig Colony for epileptics in the State of New York.

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[1] "Some European Asylums," "Amer. Jour. Insanity," July, 1887.

I will say that I believe improvement and reform are constantly going on in asylums throughout the world; that no one is more anxious than are their superintendents to make progress in the care and management of the insane. They are rapidly reaching the best methods of dealing with the insane poor. If any are tardy in this advance, it is because they are so often hampered by the never-ending overcrowding of our public asylums, by the interference of politics, by the lack of money, by the want of a sufficient number of medical assistants, and by a multiplicity of official duties.

While these statements are undoubtedly true,—and great credit is due the asylum physicians of the present day for their strenuous efforts in behalf of their charges,—I believe that the ideal treatment of almost any insane person is to be sought outside of an asylum. After an asylum experience of some years, and an experience of many years, too, in private practice, I feel that I am in a position to judge fairly well of the relative merits of treatment in and out of asylums.

Theoretically, it ought to be the right of every individual in sickness to receive the best treatment that medical science affords; but this right can be enjoyed by very few. There are too many interfering conditions. Not every injured man is within reach of the best surgeon; not every fever-stricken one convenient to the best physician; and few are the deaf, the blind, the lame, those with crippled bodies and those with disordered minds, who ever really receive the best treatment that the world can give. The intelligent doctor and the scientific skill are not the only requisites. Other conditions are good nursing, the most suitable climate, the best hygienic surroundings, the best moral atmosphere. In dealing with affections of the body solely, there is often much to be desired; but it is particularly in the treatment of those who are mentally as well as physically afflicted that so much which should be done is left undone. The obstacles in the way of securing the best treatment are multiplied in the case of the insane by the dethronement of the supreme centers of psychic function.

Just as a hospital is a better place than a tenement house for a surgical patient or a case of fever, so is the asylum superior to the home in the caretaking of the pauper and indigent lunatic. The acutely insane of the poorer classes are best treated, at present, in our large public institutions; and those among the moderately well-to-do, either at home or in the small private asylums. Only the insane of the wealthy classes can, perhaps, enjoy and carry out ideal methods of treatment in their own homes, in country houses, or in foreign travel.

It is, of course, needless to say that there are many degrees of insanity; that there are hundreds of cases that are never obliged to go to an asylum at all; that in society are many insane persons carrying on legitimate occupations and caring for themselves and families; and that, on the other hand, there are cases for which nothing but commitment to an asylum would be suitable or feasible. But we should not send any patient to an asylum unless he needs restraint because of danger to himself or others, or because proper treatment and supervision are difficult in his home, owing generally to poverty or other insurmountable conditions. The sooner a case of acute insanity occurring in a pauper or an indigent is removed to an asylum, the better are his chances for recovery. This merely signifies that the earlier treatment is undertaken by those who are familiar with the management and care of the insane, the better for the patient. Early treatment by physicians of experience in psychiatry is demanded. At present this end is best attained by resort to the asylums of the neighborhood. But the writer has often called attention to the need of increasing and extending the facilities for the early treatment of the insane—a matter which can be accomplished in several ways. The lines of progress in such direction are:

(1) The opening of special reception-wards or pavilions for the insane in general hospitals; (2) the establishment of psychopathic hospitals in large cities; (3) the creation of outdoor departments in connection with asylums situated in densely populous districts.

Before taking up the matter of the treatment of insanity, a few words should be said as regards

Prophylaxis.—Naturally, the question of the proper care and education of children with a tainted line or lines of ancestry often comes before the physician. Much can be done to ward off impending future evils by due and early attention to the mental and physical evolution of such children. One can not begin too soon to regulate the life of these little ones. The very milk of a weak and anemic mother may diminish the feeble resistance of a degenerate child. From the day of birth the prophylaxis must begin. The points to be observed in the effort to accomplish this are as follows:

1. Cultivate the body of the growing child. Develop him physically by careful and regular diet, regular hours of sleep, outdoor life, efficient systems of exercise.
2. Let his training be muscular rather than intellectual, manual training rather than lessons, especially in the early years of childhood. No schools until the age of seven or eight years.
3. The child with degenerate tendencies should be forbidden all nervous stimulants, such as tea, coffee, wines, beer, tobacco.
4. Seek to develop the resistance of the organism to all external stimuli, hardening his body by the daily morning cold bath, frictions, exercise, a hard bed, a cold sleeping-room; accustoming his mind to the courageous endurances of pain and mental stresses.
5. Guard well the epoch of puberty.
6. Let the occupation chosen for later years be also one for the

muscles rather than for the mind, an outdoor rather than an indoor calling, a country rather than a city life.

Isolation.—On being called to see a patient suffering from insanity, the first point which arises is whether he should be sent to an asylum or not. This is generally a question of means. Isolation from the immediate friends is in nearly every case a requisite. If the patient belongs to the indigent or to the middle classes, isolation and the best treatment for his malady are only to be satisfactorily obtained in an asylum or hospital for the insane. Among the well-to-do, the needed isolation may be successfully secured in his own house, in an ordinary sanatorium, or by means of travel with a suitable nurse, companion, or physician. The kind of treatment best adapted to the nature of the case must be decided by the physician. The quiet of a private house in the city or country is best for some cases, while the tonic and stimulus of foreign travel are indicated in others. It may be stated that, when travel seems to be the prescription required, the greater the change from the environment in which the mental disorder developed, the better. The cities of Great Britain and the Continent do not differ essentially from our own cities, and patients should not be sent to such places with the idea of securing a change of environment. Norway in summer, Egypt in winter, and Mexico in either summer or winter, are regions which offer the greatest inducements in the way of tonics to the nervous system and stimulus to the mind, and all three are, at the same time, peculiarly restful and calmative.

If these methods of home, country house, or travel are for any reason impracticable, then the smallest private asylum that can be found is to be selected, for the fewer other insane persons and the greater number of sane persons the patient comes in contact with, the better will be his chances for recovery. There is a need for physicians in practice in the country who will be duly authorized and empowered by law to receive in their own homes and care for one such patient. The chief drawback in home-treatment, if long continued, is usually the bad effect of association with an insane person upon other members of his family, particularly if they be neuropathic. With a sufficiency of nurses and room, there is no contingency in the treatment of the insane that can not be guarded against. These being provided, the worst features in a case, such as violence, homicidal and suicidal tendencies, attempts at self-mutilation, etc., may be as well avoided outside as inside of an asylum. There are cases in which—though I am opposed to mechanical restraint in great measure—I should employ long-sleeved night-gowns, or even camisoles, rather than let them go from home before all means of cure had been tried at least for a few weeks' time.

The conditions and propensities that we have to combat are many. The choice of method must be the result of careful deliberation, and after judicial survey of all the features presented. We usually need the assistance of skilled and experienced nurses. Thanks to the asylum training-schools, there are numbers of such trained nurses of both sexes to be had in our large cities.

TREATMENT OF ACUTE CASES.

In acute cases, whether of mania or melancholia, it has been my experience that confinement to bed is a valuable factor in cure. Hence, on being called to such a case, I have the patient put to bed. Due precautions are taken as to the removal of all sharp instruments, weapons, drugs, cords, door-keys, and the like, and by a simple device the windows so arranged that they may not be opened beyond six inches; otherwise the furnishings may be left as they are without attention.

Insomnia and mental and motor excitement most frequently demand our best skill. In emergency, I am in the habit of using duboisin sulphate hypodermatically in the dose of $\frac{1}{100}$ of a grain, or sometimes hyoscyamin, or hyoscin hydrobromate in doses of from $\frac{1}{100}$ to $\frac{1}{60}$ of a grain hypodermatically, though these latter are not so satisfactory as duboisin. But for routine treatment of insomnia and maniacal excitement I much prefer hydrotherapy to drugs. In some cases the prolonged warm bath (70°–90° F.) for from one-half to two hours may be used, but in all cases the hot wet-pack is applicable. Sometimes when the wet-pack does not suffice to quiet fierce maniacal excitement, I use duboisin in addition, or give doses by the mouth of paraldehyd, trional, and sulphonal, all of which are valuable hypnotics.

In acute depressed conditions, on the other hand, opiates usually act best in cases in which hydrotherapy does not subdue the insomnia, distress of mind, and disordered nervous system. Among opiates, codein seems to offer advantages over others, and the contraction of a habit need not be feared. The aqueous extract of opium or morphin may be given hypodermatically.

The refusal of food is another element of danger. Acute insanity, besides rest in bed, quiet, and repose, needs overfeeding to balance the great waste of tissue going on in the system. While many cases of acute mania will eat and drink ravenously at times, from the nature of things their actions are uncertain, and the nurse should be instructed to feed the patient almost hourly and keep account of what is given. Milk, raw eggs, meat-juice, and occasional stimulants must, in extreme cases, be our chief reliance. Having an intelligent and assiduous nurse at hand, the necessity of feeding with a tube will only rarely occur. When required, the soft rubber stomach-tube may be introduced by the physician through the mouth or nose, a funnel attached, and the liquid mixture of the substances named allowed to flow in.

There are cases (some of the insanities of puberty and adolescence, and other forms) in which anaphrodisiacs modify distinctly the trend of delusions. There are cases in which intestinal antiseptics achieve noteworthy results; indeed, the instances are few in which attention to morbid states of the alimentary canal is not rewarded by considerable benefit to the mental condition of the patient. Arguments with patients upon delusions, more or less fixed in character, often has, despite the opinions of numerous alienists to the contrary, decided value in altering their beliefs, and at times even eradicating their insane ideas altogether. It is true that occasional argument is generally of no avail. Such moral treatment must be sedulously and perseveringly employed, daily and for weeks or months, to insure success. Argument is a species of suggestion. The tactful and judicious physician will not make use of it in cases where it leads to irritation and would seem to be injurious.

The most important remedial agents employed in insanity are as follows:

The Rest-cure.—This has already been briefly referred to. It was in 1860 that Hilton began his series of lectures on rest and pain, in which he pointed out how much rest had to do with growth and repair of the bodily tissues, and fifteen years later Mitchell wrote of the value of rest in the treatment of hysteria and neurasthenia. Nowadays, however, we apply the principle of rest to a great variety of nervous disorders. Besides its indication in many cases of hysteria and neurasthenia, we find it of the greatest benefit in all sorts of nervous and mental troubles, and especially in such as evince a tendency to waste of tissue and to exhaustion.

Most cases of acute mania need to be treated by rest, which should be made as absolute as possible. Many cases of acute melancholia recover more quickly when confined to bed. While in many mental cases the rest should be absolute for a period of several weeks in order to insure a successful termination, it is astonishing how much benefit can be obtained by a modified rest treatment—that is, by merely prolonging the daily amount of repose in bed. The principle is to apply rest methodically, and in proportion to the degree of nervous exhaustion, strain, or irritation.

When rest is made nearly absolute, it is necessary that tissue metabolism should be encouraged by attention to the amount and quality of food, and especially by substitution of some passive artificial exercise for the active movements upon which the organism has hitherto depended. This is accomplished chiefly by massage.

Massage.—Massage was a favorite remedy and luxury in ancient Roman times, when it figured as the Aliptic Art;[1] so that it is not at all a new remedy, but its vogue in recent years has assumed enormous proportions, and it has received a scientific study and systematization to which the ancients were strangers. This rubbing, beating, and kneading of the trunk and limbs, when skilfully done, is an essential adjunct to the absolute rest treatment. It is invaluable in many kinds of pain, and it often surpasses drugs as a soother of irritation and an inducer of sleep.

Diet.—It is needless to say that in connection with a form of rest treatment simplicity should be the rule as regards food. The selection should be made from the point of view of easy digestibility, and foremost in this regard stand milk and its various preparations. Where milk can not be taken in its ordinary form, some more digestible preparation may be employed, such as peptonized milk, koumiss, matzoon, or somal. In cases undergoing a rest treatment this is the main staple of food, and it should be given frequently and in considerable quantity. Overfeeding is indeed another principle in the treatment of any of the nervous and mental diseases in which exhaustion is a feature. Thus, absolute rest and overfeeding must be our chief reliance in acute mania, and in severe types of melancholia. Many cases require feeding every hour or two hours. Raw or soft-boiled eggs, rare or raw beef, specially prepared cereals, and sometimes green vegetables and fruits may be added to the diet. (By specially prepared cereals I mean simple boiled rice, stale bread in the form of toast, or, better, bread which has been twice baked—Zwieback). Stimulants are only occasionally indicated, and then especially in acute maniacal or other dangerously exhausting conditions.

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[1] "The Aliptic Art: a Historical Study," by the author, "Phil. Med. News," Aug. 11, 1883.

A somewhat similar form of diet is appropriate for mental disturbances having a rheumatic or gouty diathesis as a basis. The same diet is essential in all cases of insanity, neurasthenia, epilepsy, and so on, which seem to depend upon auto-intoxication from fermentative or putrefactive changes in the intestinal contents, and such cases we find nowadays to be not at all infrequent.

Hydrotherapy.—When in 1893 I wrote a paper on "Hydrotherapy in the Treatment of Nervous and Mental Diseases" ("Amer. Jour. of the Med. Sciences," February, 1893), there was really no place in the city of New York to which one could send patients and have his own ideas as to treatment faithfully carried out; nor did I know of a single asylum for the insane in this country installed with hydrotherapeutic apparatus, such as I had seen in a number of asylums abroad, even in so remote a country as Greece. Now I could name many public and private asylums which are equipped with arrangements for this purpose.

Water affects the nervous system in a variety of ways.

Cold baths increase and warm baths diminish the irritability of the brain and spinal cord in a reflex manner by stimulating the sensory and vasomotor nerves of the skin, thus influencing the cerebrospinal circulation.

Short cold baths, especially when combined with sprinkling, showering, or rubbing, are powerfully stimulating, exhilarating, and tonic. Cold baths stimulate peristalsis and the visceral reflexes in the cord, and increase blood-pressure. Prolonged warm baths, steam and hot-air baths, and the hot pack are relaxing, fatiguing, and tend to induce sleep. Warm baths diminish arterial tension and reduce the irritability of individual nerves and the whole nervous system. The spinal douche is of the greatest service in many nervous disorders, because of its remarkable tonic, revulsive, and derivative effects. It is a powerful mental as well as physical stimulus. By means of various nozzles it is ejected in the form of a strong stream up and down the back of the patient for a few seconds only, at a distance of some ten feet. Patients with good reaction do not need any special preparation, but at the beginning it is well to have the patient take a warm bath or stay a few minutes in a hot-air box previous to its application. At the first séances the water should not be too cold. Later, it may be gradually lowered to 50° F. It should be taken every day, when possible. Occasionally this cold spinal douche is alternated with a hot douche (the so-called Scotch douche). This is an exceedingly successful procedure in many cases of hysteria, neurasthenia, and in lethargic and hysterical forms of insanity, where there are sluggish intellect, great depression, apathy, stupor, catalepsy, etc., and in any case of nervous and mental disease where anemia, chlorosis, or gastric trouble exists.

In insomnia there is no other remedy so generally efficient and at the same time so innocuous. I have seen it successful in wakefulness from every kind of cause, and in cases seemingly intractable to other remedies. There are two hydriatic procedures for the production of sleep. One is the prolonged warm whole bath, at a temperature of 70° to 90° F., for from one-half to two hours just before retiring. This is indicated in mild cases of insomnia. But the hot wet-pack is more effectual and more widely applicable in all forms of sleeplessness, whether in nervous or insane individuals. It is applied in this way: A blanket, nine by nine feet, is spread upon the patient's bed, and upon this a sheet, wrung out dry after dipping in hot water, is laid. The patient lies down upon this, and the sheet is at once evenly arranged about and pressed around the whole body, with the exception of the head, after which the blanket is also immediately likewise closely adjusted to every part of the patient's body. Other dry blankets may now be added as seems necessary. The patient remains in this an hour or longer; all night, if asleep.

I know of no better treatment of acute maniacal conditions, for instance, than rest in bed, overfeeding, the hot wet-pack, and the occasional employment of some sleep-producing agent.

Treatment of Auto-intoxication.—Researches in the physiological chemistry of digestion, as well as observations in many pathological conditions, have established that auto-intoxication from the absorption of poisonous substances generated in the alimentary canal by putrefactive and fermentative processes is not only a real thing, but a frequent factor in the etiology of a number of nervous disorders, such as headache, neurasthenia, hysteria, neuralgia, and even graver maladies, like epilepsy, melancholia, mania. It behooves us, therefore, in these diseases, to investigate carefully for evidence of any such cause. Periodical or constant attacks of gaseous diarrhea are somewhat indicative of this condition. Frequently the condition of the bowels furnishes no information of the actual state of affairs. Recent researches tend to show that an excess of ethereal sulphates in the urine (indican) in connection with other symptoms is a good index of auto-intoxication.

When auto-intoxication is suspected as the causative factor in any nervous disorder, it is essential to regulate the diet in the manner already mentioned, and there are at our disposition a number of intestinal antiseptics which, though not always efficient, are yet often of very great benefit. I have found, in my own practice, that beta-naphtol is one of the best intestinal antiseptics. I give it in capsules of five grains each, two hours after eating, with water. In several cases of epilepsy and of melancholia it has acted exceedingly well. In many cases of epilepsy salicylate of soda has also proved itself of great value. Salol, too, is a good intestinal antiseptic. Sometimes I have made excellent use of peppermint for the same purpose. I think the abundant use of water a necessary adjunct in the treatment, usually advising the drinking of hot water several times daily on an empty stomach, and sometimes adding thereto frequent flushing of the large intestine with warm water.

Electrotherapy.—General faradization with a current sufficiently strong to contract the muscles has much the same value as massage where the rest-cure is employed; it exercises the muscles and stimulates metabolism. Over and above this it has a tonic effect. Galvanism is only of use in complicating conditions, such as neuralgias, sciatica, and the like. The same is true of the static and sinusoidal currents. Electrization of the head for the purpose of influencing illusions, hallucinations, and delusions is occasionally of service, but doubtless its influence is almost wholly of a suggestive nature. However, it is not to be interdicted on that account, for suggestion is in itself a valuable therapeutical adjunct, and so good a method of increasing its usefulness as is afforded by electricity is not to be slighted. Suggestion is a species of psychic therapy.

Drugs.—The narcotics are of great importance in the treatment of insanity. Among these, opium and its alkaloids easily stand first.

Opium, morphin, codein, all have a hypnotic effect, but their especial value lies in their sedative influence upon mental hyperesthesia, anxious states, etc.; in their contraction of the blood-vessels, and in their stimulation of the nutrition of the central nervous system. The hypodermatic use is best. They are particularly indicated in melancholia, acute alcoholic psychoses, and hallucinatory paranoia, very seldom in maniacal states. They are contraindicated in most maniacal conditions, collapse, fatty heart, uncompensated valvular disease, and marasmus. The patient should not know the name of the drug used. Opium and codein are preferable always to morphin, because of less danger of forming a habit. The doses must be gradually increased. The constipation at first present during the administration of opiates disappears later.

Hyoscin, hyoscyamin, and **duboisin** are isomeric alkaloids, and have much the same qualities and are alike in their effects upon the organism. Next to the opiates they form the chief drugs of the alienist's armamentarium. Their great value lies in their sedative influence upon motor centers. They are used hypodermatically in doses of from $\frac{1}{100}$ to $\frac{1}{16}$ of a grain. Almost immediately after injection the muscles become incoördinated and weak, and in ten or fifteen minutes the patient sinks into a light slumber which lasts from six to eight hours. The peripheral arteries are contracted, giving the patient a striking pallor; the breathing is slowed, the pulse retarded or made intermittent, the throat rendered very dry, and the pupils enlarged and accommodation paralyzed. These drugs are contraindicated in heart disease, and in no case should they be continued any length of time.

Precious as they are on the right occasion, their employment should be subject always to the careful and judicious supervision of the physician.

Another feature of their physiological action to be borne in mind is their power to induce dreadful hallucinations in a well person—a fact which emphasizes the need of care in administering them to an individual whose mind is trembling in the balance. Long-continued use of these alkaloids interferes with nutrition.

From what has been said of the action of these drugs, it will be seen that their effectiveness is most manifest in conditions of motor excitement, in mania, agitated melancholia (combined with morphin), in agitated dementia, and in the motor excitement of epilepsy or paresis. I have often been able to feed excited patients who refused food, immediately after the injection of the alkaloid, during the few minutes that elapse before the advent of sleep.

The **bromids**, aside from their particular value in epileptic psychoses, are often useful in other forms of mental disease, owing to their effect in diminishing cerebral activity and reflex irritability. In epileptic insanities the combination of the bromids and opium is especially effective. They are of use in any mental excitement which is conjoined with some reflex irritability (illusions and organic sensations, uterine and genital disorders). As an anti-aphrodisiac they are employed in insanity with erotic manifestations. In large doses, sixty to ninety grains and over, they act well as a safe and innocuous hypnotic.

Chloral hydrate is not so much used as formerly, though its hypnotic effect resembles very closely natural sleep. It is applicable to acute hallucinatory conditions, insanities associated with chorea, and in the epileptic psychoses. In status epilepticus, per rectum it is one of the most valuable remedial agents. In some conditions, combinations of chloral with morphin are of much utility. Chloral is a heart poison, and its use is contraindicated in cardiac and vascular disease. Chloralamid is of little value.

Paraldehyd is a simple hypnotic whose utility is not sufficiently appreciated. Naturally, its bad taste and the rather disagreeable odor left upon the breath have limited its sphere of usefulness; but it has no bad influence upon the heart or nutrition. It can be given in heart disease, and patients seem to thrive and grow fat upon it. The dose is from one-half to two drams, but increasing doses are necessary, and I have had patients who have taken four or more drams at a dose. It is especially useful in conditions of inanition and in insanities founded upon hysteria or neurasthenia. The taste and odor of the drug can be concealed in orange-water or weak brandy. Amylene hydrate is of less value; it stands between chloral and paraldehyd.

Trional and **sulphonal**, as simple sleep-producing agents, are preëminent where nothing but sleep is the object to be attained. Trional acts quickly, sulphonal slowly; hence a combination of the two in equal doses is particularly fortunate in its results, inducing, as it does, rapid and prolonged slumber. Five to ten grains each, or more if indicated, may be given at bedtime with a glass of hot milk. The tastelessness of these drugs affords the possibility of administering them without the knowledge of the patient, mixed with salt or sugar, or spread with butter upon bread. Sulphonal used for a long period produces muscular weakness and incoördination. Both of these agents may, after a time, give rise to some disorder of the alimentary canal. They are said to occasionally increase the intensity of auditory hallucinations.

Moral Treatment.—Psychotherapy is among the most important means of treatment of insanity. The general practitioner is especially concerned with it in the early stages of mental disorder; later, if the patient is turned over to the care of the asylum, it is still of the utmost importance, and the physicians in institutions know well the necessity and utility of moral agencies in effecting a cure or in at least ameliorating the condition of their charges. Physicians who have much to do with ordinary functional nervous disorders—hysteria, neurasthenia, mild depression, and hypochondriasis—are familiar with the wonderful influence they are able to exert over the mental attitude of patients thus afflicted, by kindness, patience, firmness, interest, and sympathy. Everything they say or do, if rightly said or done, conveys a suggestion, inspires hopefulness, increases the efficacy of their prescriptions, points out the way to health and a new lease of life. The insane also are in the same way dominated by the personality of the physician and of those chosen to carry out his instructions. Some physicians are fortunate enough to possess peculiar gifts in this way, and their influence is potent for incalculable good. Aside from this personal influence, the physician is called upon to direct and regulate the entire disposition of the time of the patient and to make for him the environment suitable to his malady. He prescribes isolation from friends, the care of strangers, the rest-cure, the periods and kinds of exercise, the mental and manual occupations, the amusements, all of which go to make up psychotherapy. Some of the principles of this moral treatment we will now briefly touch upon.

The value of isolation in melancholia and of the rest-cure for both acute mania and melancholia has already been mentioned. There are cases of melancholia, however, in which a modified rest-cure is better than the complete rest-cure. In such cases, after recumbence in bed from six in the evening until noon the next day, much of the afternoon may be spent in simple exercise, such as walking slowly about out-of-doors. It is best not to seek, by amusements, visits of friends, and other cheerful devices, to raise the melancholiac from his depression, for usually these attempts rather add to his misery by force of contrast. A neutral atmosphere, so far as the emotions are concerned, is best, though an occasional word of confident reassurance is useful.

In acute stages of insanity it is best not to discuss the hallucinations and delusions of the patient, although neither physician nor nurse should ever fall in with or act upon his erroneous ideas. Whenever good judgment suggests, a brief but positive denial of the truth of the imaginings of the patient should be made. Later on such correction may with advantage be made more frequently and constantly.

When the patient is not taking a rest-cure, occupation of some kind is essential to his progress toward recovery. Most useful are all forms of muscular or manual employment, for labor of this kind keeps the attention more or less fixed upon what is being done, the flow of ideas is checked and limited to a considerable degree, and the mind is prevented from concentrating itself upon illusions, hallucinations, and delusions. Moreover, muscular exercise is an outlet for superfluous energy; motor excitement is reduced by it; tissue metabolism is accelerated; and when the work is over, the organism gains all the more readily a certain composure of mind and repose of body. Out-of-door occupation is best—garden and field work for men, garden work for women; walking, bicycling, etc., for either sex. Among indoor employments we have ordinary housework, drawing, knitting, sewing, embroidery, carpentry, wood-carving, etc., all of which employ the muscles methodically. In certain cases mental occupation is useful, though it should be of the simplest kind. For instance, during my practice at the Hudson River State Hospital for the Insane, we found much value in the establishment of a regular country school, attended by patients of all ages. We had "spelling bees," copying lessons, reading aloud, blackboard exercises, geography, simple arithmetic, singing, and so on.

A very important point in the management of the insane is never to practise deception upon them in any way. Be absolutely truthful in every statement to them. Never remove a patient to an asylum under the impression that it is a hotel or sanatorium. It is better to state exactly what is going to be done, and then use force in the removal, if necessary.

Hypnotism has been frequently practised upon the insane, in the effort to modify hallucinations or delusions, rarely with any definite success, occasionally with ill results, and generally with no effect whatever.

There are a few conditions among the insane which require particular treatment or management. Among them are:

Suicidal Tendencies.—Suicidal patients are among those who require constant watching and the removal of every means of self-injury. This is often difficult in treating such patients in their own homes. How difficult, it may be conjectured from the fact that, even in asylums, with all their safeguards, suicide is by no means infrequent. Thus, forty-eight patients in the asylums of the State of New York committed suicide between October 1, 1888, and September 30, 1896.

Suicidal patients are to be watched night and day, and kept in bed, and even put in restraint, if desperate. I have known a patient to strangle herself with a cord while lying in bed under the eye of a nurse. Another, broke a small piece from a china plate and tried to cut her wrists under the bedclothes. While suicide is most common among melancholiacs, patients with general paresis, paranoia, epileptic psychoses, and toxic delirium sometimes attempt it. The physician attending such patients should see to the guarding of windows and the removal of keys, hooks, scissors, weapons, drugs, strings, long pins, matches—in fact, of all instruments and means which he may suspect to be utilizable for a suicidal purpose.

Refusal of Food.—The acutely maniacal often can not be made to take sufficient nourishment, because they do not stop long enough in their ideomotor excitement to permit of eating. The watchful and persevering nurse can generally, by persistent effort, induce the patient to swallow a considerable quantity of liquid food (preferably in a metal or heavy china cup, because the patient frequently knocks the vessel from the hand of the nurse). Such patients can often be fed, as already stated, immediately after a hypodermatic injection of hyoscin or duboisin before the supervention of sleep.

Other patients refuse to eat because of delusions of poverty or poisoning, suicidal proclivity, or simply from absolute distaste.

Where ordinary means fail, the nasal tube should be resorted to,—one of large caliber with rubber funnel attached,—and through this, once or twice daily, a mixture of a pint of milk, two or three raw eggs, a little meat-juice, and, if needed, brandy, may be introduced.

Before resort to this means nutritive enemata may be employed (three raw eggs, a half-pint of milk, a half-pint of water, and a little meat-juice).

I have been in the habit of delaying the use of the nasal or stomach-tube to the last moment of safety, even for several days, rather than subject the patient to the excitement of its employment. It is only in rare instances that feeding is not effected in some other way before the use of the tube becomes imperative.

Violence and Destructiveness.—Hypodermatic medication and hot wet-packs are indicated in periods of excitement with tendency to violence and destructiveness. It has already been intimated that active physical labor or exercise is a safety-valve for patients with proclivities of this kind. Isolation in an empty room with protected windows is sometimes resorted to in institutions, and abroad the padded room is a favorite place for patients whose violent jactitations may lead to serious injuries to himself. The padded room consists simply of a room lined as to walls and floor with cushions. Mechanical restraint is used in the last extremity, when chemical restraint and other means have failed. The camisole and safety-sheet are employed only in cases with desperate suicidal tendencies, proclivity to excessive masturbation, great violence and destructiveness, and where needed to keep in place surgical dressings, splints, etc. In asylums mechanical restraint has been nowadays almost entirely abandoned.

Masturbation.—Masturbation is more often the consequence and concomitant of insanity than its cause. It may be ameliorated occasionally by drugs like bromids, camphor, and lupulin. Cold baths and hard physical labor are more successful in combating this habit. In excessive masturbation, constant watching day and night or the use of mechanical restraint is necessary. The use of blistering fluids on the genital organs is only of temporary service. There are instances in which the habit is so fixed and so uncontrollable—for example, among some imbeciles—that surgical interference would be quite justifiable (castration, clitoridectomy, ovariotomy, section of the pudic nerves, ligation of the vas deferens).

CHAPTER VI.

MANIA.

Definition.—Mania is a form of insanity characterized by emotional exaltation, acceleration of the flow of ideas, and motor agitation. It is probable that the elated mood and the hyperexcitation of intellectual processes are both primary and simultaneous in their development. The motor excitement results from the conversion of the swiftly flowing ideas into acts.

Etiology.—There is no special etiology for mania—what has been said in the chapter on General Etiology has application to this form. It may be said, however, that mania is ordinarily a disorder arising between the twelfth and twenty-fifth years; that it is more common in females than in males; that individuals of sanguine temperament are most liable; and that it is, upon the whole, rather an infrequent type of insanity. Hereditary taint is found in seventy-five per cent. and degenerative stigmata in twenty per cent. of cases. The percentage is larger for the periodical form.

Mental Symptoms.—An outbreak of mania is preceded by a period of depression lasting from a few days to a few weeks, sometimes as long as two months. This prodromal stage is characterized by a general feeling of malaise, vague uneasiness, and hypochondriacal complaints, accompanied often by headaches, cephalic paresthesias, constipation, loss of appetite, sleeplessness, and some loss of flesh.

When the true mental disorder begins to manifest itself, the sorrowful mood begins to give way to an exalted condition, which the patient looks upon as a state of renewed health and well-being. He takes a renewed interest in everything, and becomes unusually cheerful and talkative. The degree of increasing exaltation varies much in different cases. In mild cases the patient begins to surprise his intimates by his loquacity, facetious remarks, jocularity, and by his rather immoderate actions and undertakings. He enters upon many new schemes; makes innumerable calls upon friends and acquaintances; writes numberless letters; purchases unnecessary articles; and is inclined to excessive indulgence in tobacco, wine, and venery. There is considerable mobility or lability of the emotions, so that the elation may readily pass into conditions of anger or tears over trifles. In more severe types all of these symptoms are aggravated. A veritable chaos of ideas throngs through his mind, and the effects upon movement of this crowding series of ideas amount to a constant motor agitation. The patient laughs, declaims, sings, shouts, makes grimaces, dances, runs about, and becomes destructive and filthy, all inhibitory idea-associations ceasing to have any influence over the rioting torrent of thought. In still severer grades we have the picture of an acute delirium, boisterous incoherence, a motor agitation attaining to violent jactitation, and an actual and considerable increase of temperature.

The patient with mania is fundamentally optimistic and egotistic. Everything about him is rose-colored. He feels rejuvenated; rejoices in his health, strength, and vitality; is delighted with the vivacity of his ideas and the untrammeled virility of his intellectual processes. His general and special sensibilities are ordinarily unaffected; in only about one-fifth of the cases are illusions and hallucinations present, and these are almost always limited to vision. Occasionally there are illusions and hallucinations of taste and touch. Illusions of the special senses are more frequent than hallucinations. The manias of extreme youth or age and alcoholic mania are especially prone to manifest hallucinations. Mania marked by the presence of numerous illusions and hallucinations is often designated as hallucinatory mania.

The accelerated flow of ideas in mania is naturally most conspicuous in the speech of the patient, which varies from garrulity to logorrhea. In the milder degrees of loquacity we are still able to follow the sequence of associations. The sentences are often bound together by the ordinary relationship and connections of ideas, but among which many latent ideas spring into consciousness and expression; and, again, the sounds of words spoken suggest others of similar sound, giving rise to rimes and assonances. Thus, the sight of the physician may suggest drugs, a certain apothecary, in a special street, in some familiar town; and the town may in turn give rise to another series. On the other hand, the physician's "How do you do?" may invoke a string of assonances (verbigeration) commingled with sentences expressing their associated ideas—shoe, two, new, grew, blue, crew, etc. But in the more striking grades the logorrhea is so pronounced that it is impossible to find clues to any association, whether of sound or idea. It becomes a chaos of words, consequent upon an actual dissociation of the ideas in the rushing stream of thought—a secondary incoherence. The entire loss of inhibitory control of ideas is especially shown in the absolute lack of modesty, in the tendency to the employment of vulgar and obscene words and expressions. This profanity and obscenity become all the more astonishing by contrast, when it is observed, as it often is, even in the most refined and cultured of women.

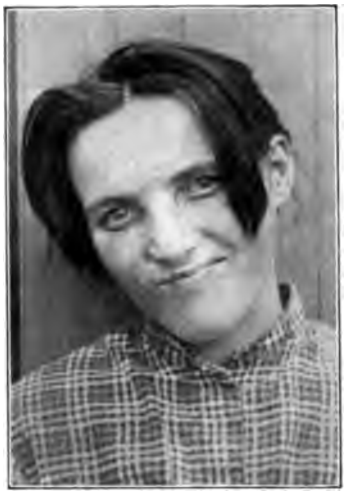

Fig. 303.—Mania (puerperal in origin).

The attention of the patient with mania is extraordinarily increased, so that the most insignificant trifle in his environment does not escape him. But this very increase of the power of attention, combined as it is with an unpausing stream of ideas, entails an absolute lack of concentration. His attention can not be held a moment. The patient's memory, too, seems preternaturally intense, and it is remarkable how, after recovery, he may remember all the details of his delirious activity with great distinctness. Indeed, the patient, in the midst of the chaotic turmoil of his mind, often recognizes, as if he stood apart from and judged himself, the very madness of his fancies and acts. The judgment-associations are, in fact, normal.

The elated mood and rapid flow of ideas give rise to delusions of expansive character, mostly in regard to strength, beauty, and intellectual powers, but often also in relation to wealth, social position, etc. In severe cases there are the most marked delusions of grandeur, the patient affirming himself or herself to be a prince, president, king, queen, Christ, the bride of Christ, the mother of God, etc. A peculiarity of these affirmations is their transitory character, their impermanence. A patient will, in the same breath, call himself a millionaire, broker, and king, and in the next a minister of the gospel and railroad magnate. If sharply told by the physician to stop such nonsense, he will often say it was only a joke, or he had said such things for fun. This shows very well the latent consciousness of the patient of the true state of affairs.

The sexual instinct is morbidly exalted, giving rise in both sexes to immodesty and obscenity of speech and manner, and often to sexual excesses and masturbation.

The actions of patients with mania correspond in character to the degree of acceleration in the stream of ideas. When this is very great, turbulence, violence, and destructiveness are common, not with any homicidal or suicidal intent, because they are incapable of acts requiring any particular concentration of mind or reflection, but simply as the result of uncontrollable automatic impulsions.

Sleeplessness is characteristic of this condition. General sensibility appears to be benumbed, probably because of the want of concentration of thought. Patients seem insensible to changes of temperature and to severe pain. Such a state often masks the most serious disorders, like pneumonia or the pains of labor. I once made an autopsy upon a woman suffering from acute mania who died suddenly. She had been for days in the wildest uproar of mind and body. The cause of death was an acute peritonitis from rupture of a perforating duodenal ulcer. The peritonitis had evidently existed for several days, yet this painful affection had clearly had no effect upon the course of the mental and motor symptoms.

Some cases of mild maniacal character exhibit a peculiar tendency to logically explain and excuse their insane acts, and this type is often designated as reasoning mania.

As already stated, mania begins with a prodromal stage of depression. After the exalted stage has culminated and at the beginning of convalescence, a reactive stage of depression is presented, characterized by irritability, sensitiveness, and lacrymosity. This stage of depression may be so intense as to be an actual melancholia of simple nature or accompanied with stupor. In instances of this kind the possibility of the patient's having a circular form of insanity, instead of a simple mania, is to be considered.

Physical Symptoms.—Except in the severest type of mania (acute delirium) the bodily temperature runs a normal course, sometimes even showing a subnormal character. In acute delirium the temperature may reach 104° or 105° or more. The pulse is small and normal, or but slightly increased in frequency in mania. There are no paralyses, no true anesthesias. The absence of fatigue is often surprising. The deep reflexes are exaggerated, as a rule. The salivary secretion is frequently increased. Perspiration is diminished and sometimes transformed in character, so as to give a peculiar and often extremely disagreeable odor (kakidrosis). Gastric disorders are nearly always manifested, and the tongue is heavily furred, frequently dry. In severe cases albuminuria, propeptonuria, and hyalin cylinders are frequently found. The general bodily weight diminishes during the progress of the disease, but rapidly increases with convalescence. There is a corresponding condition of the appetite, an anorexia during the early stage and until the culmination; then an increase of appetite amounting often to bulimia. The face is sometimes slightly suffused, but, as a rule, marked by a yellowish pallor. As the patient emaciates this becomes more noteworthy, and at the same time the features become pinched and sharp and the eyeballs sunken. This outline and color of the face, with a tendency to dryness of the lips and a heavily furred and dry tongue, are indications of the progress of exhaustion.

Varieties.—According to the intensity of the manifestations, upon the basis of the course of the disease, from the nature of certain concomitant symptoms, and, fourthly, in relation to some of the etiological factors, mania is frequently divided into several varieties bearing some special qualification. I have already alluded to mild and severe degrees of mania,—*mania mitis*, *mania gravis*, and *acute delirious mania*,—which shade off gradually with many intermediate stages from one into the other. I have also mentioned *reasoning* and *hallucinatory mania*, which owe their names to peculiarities in the symptoms. It is customary to speak also of *acute*, *subacute*, and *chronic* mania, the first two designations referring especially to the rapidity or slowness of onset, the last qualification to the duration of the insanity in its maniacal form for a year or many years. The word chronic does not mean incurable, for cases of chronic mania of long standing not infrequently recover. The term *transitory mania* was formerly employed to describe a delirious condition of very brief duration, a few hours or a day or two, but these cases do not really present the characteristic symptoms of a true mania. *Periodic mania* is a form in which attacks of mania follow one another with perfectly normal but generally irregular intervals of days, months, weeks, or years. The attacks themselves last from a few days to a few months. Usually the prodromal depressive stage is absent, the culmination rapid, and convalescence seldom marked by the interesting depressive affects of ordinary mania. The periodic attacks are very apt to be distinguished by the presence of special symptoms, such as a reasoning tendency, tendencies to impulsive acts, arson, stealing, assaults, sexual and alcoholic excesses, and to severe headaches. The longer periodic mania endures, the less distinct become the normal features of the intervals. *Recurrent mania* and *intermittent mania* are only other names for periodic mania. Various etiological factors give rise to such designations as epileptic, alcoholic, morphin, puerperal, senile mania, etc., in some of which the mania takes a special color from its cause. Thus, the toxic manias are generally acute hallucinatory conditions.

Pathology.—The most careful investigations of the central nervous system have thus far discovered no pathologico-anatomical basis for mania. The theory still prevails that there is a condition of congestion of the higher brain-centers underlying the manifestations of mania, but this theory lacks the support of observed facts. We are, therefore, constrained to look upon the disorder as functional in its nature, as due to a morbid change in the nutrition of the cells, in the way of deficient or perverted metabolism.

Course of the Disease.—Mania terminates in recovery, death, secondary dementia, secondary paranoia, or chronic mania.

Recovery takes place in some seventy per cent. of cases. Sometimes it is exceedingly rapid, but usually the progress is gradual and rhythmical to the normal state. This rhythm is a sort of oscillation between good and bad days, but with constant improvement. Occasionally the patient improves steadily and uninterruptedly until recovered. Recovery is sometimes not perfect, so that we speak of it as recovery with defect.

Death takes place in but five per cent of cases. The cause of death is sometimes exhaustion, as in acute delirium ; more often an intercurrent affection, such as pneumonia, nephritis, and the like. Heart disease and alcoholism add greatly to the danger of lethal termination.

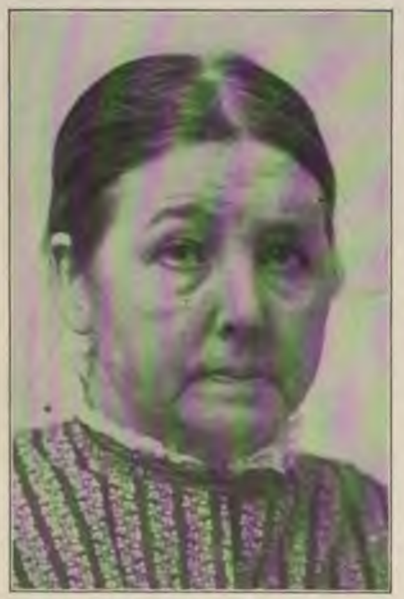
Fig. 304.—Chronic mania.

Dementia is the result of the disorder in about one-tenth of the cases. The degree of dementia varies from a slight diminution in some of the higher qualities and powers of the mind to pronounced mental enfeeblement with vestiges of the antecedent mania and complete confusion and incoherence. The vestiges of the antecedent mania are commonly in the form of motor agitation, and occasionally hallucinations and rudimentary delusions of exalted character.

A *paranoia* secondary to mania is met with in rare instances, perhaps once among a hundred cases. In such termination we observe a tendency to the systematization of some of the original maniacal delusions.

Chronic mania is quite as rare a condition as secondary paranoia. By this term is meant a continuance of the typical maniacal symptoms for a long period of time—a year or many years.

It must be remembered, too, that an attack of acute or subacute mania may be merely the beginning of a periodic mania or of a circular insanity.

Diagnosis.—One must be careful not to confound delirium from fever with an attack of acute mania. Except in acute delirious mania, the absence of fever in the mental disorder should be distinctive. The three cardinal symptoms of mania should be kept constantly in mind—viz., the exalted mood, the accelerated flow of ideas, and the motor excitement. We must determine whether these are primary or secondary to hallucinations and delusions, and whether the syndrome is complicated by other conditions, such as general paralysis and alcoholism. A very mild degree of mania may pass unrecognized, unless it is possible to diagnose it from a pronounced change in the character of the individual and from the accompanying insomnia. The physical symptoms and the defect of intellect should suffice to distinguish the exalted stage of general paresis from an acute mania. The delusions, too, of paresis have a peculiar monstrosity of character that differentiates them from the exalted ideas of the maniac. Sometimes, however, there will be difficulty in making a speedy diagnosis between these two analogous exalted conditions.

In acute hallucinatory paranoia we may encounter the three emotional symptoms of acute mania, but on examination these will be found not to be primary in their origin, but secondary to the hallucinations. In the epileptic type of acute hallucinatory paranoia defects of memory are distinctive.

Subacute types of mania may resemble congenital states of mental weakness, in so far as excesses, moral delinquencies, etc., are concerned. Here, too, intellectual defect and the early history will serve to differentiate the condition of congenital feeble-mindedness.

We can never determine from a single attack whether we have before us a form of periodic mania or circular insanity. It is only the succession of outbreaks and the cyclical character that can serve us here.

Prognosis.—Some of the prognostic data are apparent from what has gone before. In the main the outlook is favorable, since so large a percentage of the cases recover. After six months have passed the prognosis becomes only half as good, and after two or three years is quite unfavorable. There are exceptional instances of recovery after many years. The younger the patient, the better the chance for cure. A second or third attack may be recovered from completely, if they be merely recedival attacks; but if they indicate a periodic insanity, the outlook is unpromising.

Treatment.—What has already been said in the chapter on General Treatment is applicable here. The requisite isolation and supervision of a patient with acute mania can seldom be satisfactorily accomplished outside of an asylum, unless his means are sufficient to secure the needed nurses and suitable surroundings.

Rest in bed aids in the prevention of exhaustion, and renders supervision, care, and feeding more easy. To induce sleep and allay motor excitement, hydrotherapy and the hypodermatic use of hyoscin, hyoscyamin, or duboisin are extremely valuable. In mild cases, equal parts of trional and sulphonal are preferable to the drugs just mentioned. Paraldehyd is also an excellent hypnotic for mild cases.

Overfeeding is also an extremely important indication. Liquid and easily digested foods are to be recommended. The bowels should always be regulated. Brandy is added to the liquid food when exhaustion is imminent, but otherwise stimulants are contraindicated. Should there be danger of collapse, the repeated hypodermatic injections of ordinary salt and water (ten to fourteen ounces) over the abdomen or in the thigh are valuable. In the depressed period of convalescence small doses of opium are often useful.

CHAPTER VII.

MELANCHOLIA.

Definition.—Melancholia is a mental disorder characterized by a primary depressed mood associated with retarded flow of thought, and either motor inhibition or, in some instances, with an agitation expressive of anxiety and apprehension.

Etiology.—Heredity is encountered in fully one-half of the cases. Inheritance of the same form of psychosis is strikingly frequent as regards melancholia. Females are more often affected than males, almost in the proportion of two to one. Heredity, physical ill-health, and mental stress together form a triad of factors which are responsible for most cases of melancholia. This psychosis is observed at any age. At puberty it is commonly associated with excessive masturbation. Love-affairs, with the novel stresses incident to such emotions, may be an exciting cause in adolescence. Pregnancy, especially in primiparæ, is a not infrequent cause, owing to the emotional strains which are frequently clustered about this physiological commotion. The melancholia of pregnancy generally begins about the third or fourth month. Prolonged lactation may cause melancholia by inducing a general debility and anemia. The parturitional period of the puerperal state does not produce melancholia so often as gravidity and lactation. The psychoses of parturition are more commonly of the nature of acute hallucinatory paranoia. The melancholia of the climacteric is due to the physiological commotion incident to this period of involution and to associated ill-health and mental strains. In old age melancholia is often associated with senile involution and nutritional changes in the central nervous system from cerebral endarteritis. Homesickness is a frequent cause of melancholia. Auto-intoxication undoubtedly plays a considerable rôle in the development of melancholia. Occasionally severe forms of agitated melancholia are associated with chronic alcoholism. It is said that northern races and people inhabiting mountainous regions are especially subject to melancholia.

Mental Symptoms.—The affective state in this psychosis varies from simple dejection, in which every thought and everything in the environment of the patient has a sorrowful color, to a state of profound depression, in which the patient is either paralyzed by the dreadful nature of his concepts or thrown into a state of agitated suffering associated with marked precordial distress. There are many degrees lying between these extremes. This morbid depression is in many ways paralleled by and analogous to the conditions of normal grief in which we observe a varied behavior of different individuals under the influence of distressing emotions; some become strangely quiet and still; others, again, make noisy and agitated demonstrations of their grief. Normal grief, too, is often accompanied by sensations of choking and of sinking at the heart, which are similar but comparatively mild manifestations of the precordial anxiety and dread of the psychosis. We observe often in melancholia a rhythmic oscillation of the state of depression during the day, and frequently from one day to another. Thus, the depression is at its height in the morning (when suicidal tendencies not infrequently present themselves), being followed by a recession with another exacerbation toward night. Very often patients sleep better on alternate nights, and manifest intenser emotional depression on alternate days. In some cases, presenting what is known as the apathetic form of melancholia, the patients complain that they have no feeling at all; that they are affected neither by things cheerful nor grievous, pleasant nor painful; that they have no longer any love for family or home, or interest in anything; that they can never be sad or glad again. Sensory disturbances are often absent. In the apathetic variety there may be analgesia. Marked illusions and hallucinations are observed in only about a tenth of all cases of melancholia. Where they are present in great number, the psychosis is designated as acute hallucinatory melancholia. The paresthesias in the region of distribution of the vagus are neither illusions nor hallucinations, but they may give rise to delusions; they depend probably upon vasomotor disturbances. The melancholiac perceives and identifies ordinary and special sensations slowly and with difficulty. The peripheral stimuli of his environment go unnoticed. When hallucinations are present, they usually affect most of the senses, and are terrifying and dreadful in character. The patient sees the flames of hell, phantoms, and ghosts of dead persons; hears voices which

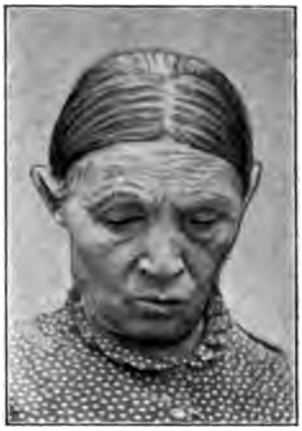

Fig. 305.—Acute melancholia passiva.

reproach and threaten him, or the sounds of machinery and other tortures which are being prepared to cut him up or mutilate him; smells and tastes horrible things, and so on.

Next to the affect of depression, the most noteworthy symptom of melancholia is the slowing of the thought processes. This is the antithesis of the accelerated flow of thought noted in maniacal conditions.

The processes of memory are retarded, and the attention of the patient difficult to gain. A minute or several minutes are required for the answer to the simplest question. Sometimes no answer is given at all, or at most the lips stir inaudibly.

The contents of the concepts may, in milder degrees, show no delusions. More often the patient attempts to explain his feeling of abject misery and distress either by the presence of some fancied physical ailment (hypochondriacal melancholia, with delusions of having syphilis, consumption, cancer, impotence, incurable disorders of the stomach, bowels, etc.), or as the result of some sin of his past life. To the delusion of having sinned an especial color is given by the character of the patient's early education. Thus, a strong religious bias gives rise to delusions of having committed the unpardonable sin, of being doomed to hell, to everlasting punishment, to be buried alive, etc. Often such delusions are connected with some trivial error of his past life. For instance, a patient of mine recently told me, "I once chloroformed a dog to death and buried him. I think now I made a mistake in not making positively sure that the dog was dead, and as a result I am doomed to be buried alive also, and to be tortured with dreadful thoughts through eternity, each day the torture growing more dreadful, up to the decillionth power of intensity."

Patients often say they are not sick, they are only wicked. They have committed sins not only against God, but against society. Not only must they undergo the punishment ordained by Heaven, but they must answer to man for infringements of human law. They are to be put in prison, to be killed, to be hung. Thus they come to delusions which are somewhat similar to persecutory ideas in that they believe the officers of the law are after them, etc. These differ, however, from the true persecutory delusions in which patients have no self-depreciatory ideas, but believe themselves to be the innocent victims of inimical conspiracies. Delusions of poverty are very common, and especially so in senile melancholia.

The conduct of the melancholiac depends upon the contents of his consciousness. In his expression we note the lines of extreme depression, or of fear and terror. The patient with the delusion of sin or poverty, for example, presents motor inhibition. He sits in one place with head bowed down, unmindful of what goes on about him, indifferent or apathetic to all questions put to him, resisting every attempt to give him food or medicine, or to dress and undress him, or to give him exercise. He is lost in the contemplation of his misery. Another patient, with these or similar depressed ideas more accentuated, or with marked hallucinations, will wring his hands, tear his hair, walk or run up and down, bewailing his misfortunes, or seeking to escape the dreadful fate in store for him. In the first case the motor inhibition may be so complete as to make the patient perfectly immobile, so that not a single voluntary movement is made; even micturition and defecation are involuntary. Such immobility is generally of flaccid character, but sometimes it assumes the phase of rigidity, a waxy flexibility, or a spasmodic resisting rigidity (catatonic rigidity). Catatonic symptoms have been noted in other forms of psychoses, but the disorder described by Kahlbaum under the name catatonia is really a form of melancholia. Suicidal tendencies are observed in every type of melancholia, but especially in those with precordial distress and agitation. In the milder

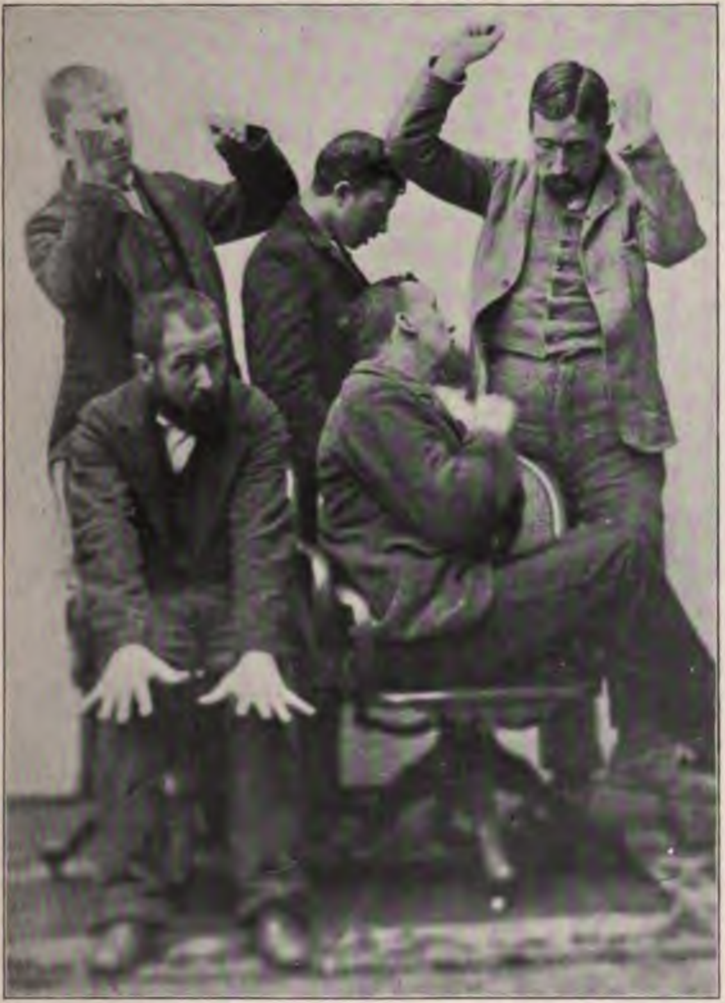

Fig. 306.—Catatonic symptoms in various psychoses (melancholia, general paresis, circular insanity, primary dementia, etc.) (photograph loaned by Dr. Atwood, of Bloomingdale).

degrees, an attempt at suicide is often the first intimation to friends of the actual existence of insanity, since in these cases, outside of the sorrowful mood of the patient, the intellectual processes may go on as before. Cases of melancholia attonita (with marked motor inhibition) also often make attempts at suicide, unexpected explosive attempts, the result of the sudden letting up of mental and bodily tension. This has been called the raptus melancholicus. Homicidal attempts and violent assaults are occasional in melancholia. A melancholy mother kills her children to put them out of an unhappy world. Or a sudden dangerous assault is made as an explosion of motor tension. Hypochondriacal melancholiacs may mutilate themselves. Patients with melancholia have also been known to enter upon alcoholic excesses to drown their misery; this is especially observed in periodical melancholia. The refusal of food is almost the rule of conduct in all forms of melancholia. Sometimes this refusal rests upon a delusional foundation: the patient thinks he can not digest his food, that it never passes through him, that he is too poor to pay for it, that he is too wicked to eat, that he must do penance, and so on. Or he refuses food with deliberate suicidal intent. Generally, profound anorexia, constipation, and gastro-intestinal disorders are at the basis of this refusal to eat.

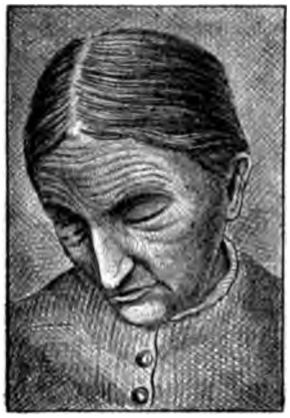

Fig. 307.—Chronic melancholia passiva.

Physical Symptoms.—The pulse is usually subnormal in frequency, though sometimes, especially in agitated forms, accelerated. The peripheral arteries are contracted and the extremities cold. The respiration is retarded and superficial, as a rule, though it may be increased in the agitated types. Sleep is much disordered, and even altogether absent, in severe cases. The patient emaciates both through refusal of food and because of disordered digestion. The gastric juice and saliva are often diminished in quantity. The tongue is foul and furred, and obstinate constipation is present. As a result of constipation, elevations of temperature may be observed, but otherwise the temperature is undisturbed. The surface temperature in the extremities is often much reduced. Amenorrhea is frequently induced by melancholia as well as by mania.

Varieties.—As in the case of mania, we distinguish *acute*, *subacute*, and *chronic* forms of melancholia; acute and subacute according to the degree and rapidity of inception, chronic from the duration.

Melancholia passiva is a term used to describe the cases with great motor inhibition of the flaccid order.

Melancholia attonita designates the type with motor tension and rigidity.

Melancholia agitata is a name used for melancholia with motor excitement.

Acute hallucinatory melancholia is the form accompanied by numerous illusions and hallucinations.

Hypochondriacal melancholia is melancholia associated with delusions as to physical maladies.

Raptus melancholicus is a phrase employed to describe the furious outbreaks of violence toward the patient himself or others, on the sudden cessation of mental and motor tension.

Catatonic melancholia,[1] already alluded to, is not a distinct type of mental disease, but simply a modification in the course of melancholia. It has often been considered as a special form of psychosis, and many alienists have argued pro and con. the question of its being a clinical entity. It is a very rare syndrome. A perfectly typical case is the following, observed by me in the Hudson River State Hospital:

CASE I.—B. R., female; age thirty-one; married, with four children; Hebrew; common education; born in United States; admitted to the Hudson River State Hospital in February, 1884; no heredity.

The first evidence of mental disturbance was in August, 1883, after the birth of her last child, which she nursed for two months, when she became sleepless, restless, and inclined to refuse food. Soon she developed the idea that she would never recover, began to bemoan her condition, and said it was hard to die so young. There was complete anorexia. She took no interest in anything, became careless of her person and dress and negligent of everything in which she had formerly been interested. Three weeks before admission she became suicidal, spoke of it, and attempted to choke herself and to cut herself with glass. She would bite her caretakers, and took every means possible to make away with herself. Her menstruation was regular. There was considerable constipation. The case was regarded as one of puerperal melancholia.

February 11th, two days after admission, she tried to beat her head against the bedstead; said some one was killing her children and putting them in a box; said arsenic was put in her coffee and that her mother was in the asylum; was sleepless and had to be fed forcibly. She became rapidly worse during the next few days; went into a condition of noisy excitement, calling for her mother, whom she believed to be in the building; mentioned her delusions of poisoning, beat and bruised herself against the bedstead, and refused all food. She was very suicidal. Her mouth and tongue became dry; she showed symptoms of exhaustion and was fed with the tube for a considerable period. She continued to refuse food, to resist all care strenuously, and to be desperately suicidal until March 15th, when she became cataleptic, with marked flexibilitas cerea; absolutely silent; noticing nothing, not even her husband, who visited her; would swallow food put in her mouth; made no voluntary motions; pulse good; bowels moved by enemata, but began to wet and soil the bed, and as she grew stronger was looked upon as rapidly becoming demented. This state of catalepsy continued, with variations from time to time, for a month or more, when she began to be destructive of her clothing, would strip herself naked, and was filthy in her habits. She remained in that condition, seldom uttering a word for months, until about the last of November, 1884, when she began to cry out loudly, "Bring me home to my children in New York. Bring me home to my children in New York," reiterating this over and over from morning until night, and accompanying the phrase with rhythmic movements of the hands and arms as if she were waving them in the direction she wished to go. There was a rhythm in the days, too, for every alternate day she was quiet in her chair and would whisper. This continued without variation for some two months, during all of which time she was eating and sleeping well and gaining in flesh.

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[1] "Catatonia," by Frederick Peterson, M.D., and Charles H. Langdon, M.D., "Proceedings of the Amer. Medico-Psychological Assoc.," Baltimore, 1897.

About the middle of January, 1885, her verbigeration took another character, the gesticulations remaining the same. She began to recite all day long, every other day, with great rapidity and with infinite variation, in rhymes of unintelligible words, as follows:

"Moccasins,"
"Voccasins,"
"Doccasins,"
"Crockasins,"
"Lockasins,"
"Tockasins,"
"Jockasins,"
"Hockasins,"
"Babies,"
"Tabies,"
"Gabies,"
"Habies,"
"Sabies,"
"Labies,"
"Mabies,"
"Kabies,"
"Nobis,"
"Gobis,"
"Jobis,"
"Chobis,"
"Sobis,"
"Pobis,"
"Tickater,"
"Fickater,"
"Sickater,"
"Lickater,"
"Mickater,"

and so on, *ad infinitum*. She only changed to another word when the possibilities of rhyme were exhausted.

She was mentally confused. When asked why she made these rhymes she said some one told her to; but this was probably an answer given because she could not explain why, for she had now no hallucinations or delusions. She was so confused that she did not feel sure it was her husband who came to see her.

A few months later she gave up the rhyming assonances and returned to the old phrase, with occasional variations, "I want to go home to my children in New York." "Won't I be glad when I get home to my children in New York." "What good times I'll have when I get home to my children in New York . . . to my cosy home in New York . . . when I get into the car which takes me to my husband and children in New York." This was the refrain for many months on alternate days, accompanied as before with rhythmic gestures of both arms in the supposed direction of New York. In the spring of 1886, on the quiet, alternate days, she began to sew. She steadily improved in flesh and was looked upon as in a state of dementia. There was no appreciable change in her condition during the summer. The verbigeration and gesticulation alternated with quiet and industrious days until the autumn of 1886, when improvement began to manifest itself in every way, and in November she was discharged as improved and went home with her husband on trial. There she recovered perfectly so that not a vestige of the insanity remains, and she is to this day in full charge of her household and family, as reported to us not long since by her husband.

In this case we have, first, an ordinary suicidal melancholia, with delusions of poisoning, the killing of her children, etc., and hallucinations of taste and hearing, and possibly sight, rapidly becoming an aggravated case of melancholia agitata of almost maniacal character, with a sudden lapse into a cataleptic condition lasting about a month, after which she was for some months silent, stupid, having to be dressed, undressed, and cared for in every way, when she began to show symptoms of verbigeration and rhythmic gestures previously described. During most of the long period presenting these symptoms she was mentally confused, but her mood was rather cheerful. She would frequently smile when any one asked her why she talked in that way, and she seemed to take pleasure in what she was constantly reiterating.

The conclusions at which we arrived in our paper were as follows:

I. Catatonia is not a distinct form of insanity—not a clinical entity.

II. There is no true cyclical character in its manifestations; hence it can not properly be classed as a form of circular insanity.

III. It is simply a type of melancholia.

IV. It is not desirable, therefore, to retain the name catatonia.

V. The term "catatonic melancholia" or "catatonic syndrome" may be usefully retained as descriptive of melancholia with cataleptic symptoms, verbigeration, and rhythmic movements, but should be strictly limited to this symptom-complex.

VI. The prognosis in melancholia with catatonic symptoms is more grave than in any other form.

VII. The treatment of the catatonic syndrome is the same as for the other types of melancholia.

Periodical or *intermittent* or *recurrent melancholia* has about the same significance as the similar designation of forms of mania.

Other names are frequently given to melancholia, such as senile, puerperal, and the like, but they merely cite some determining factor. The fundamental condition is the same.

Pathological Anatomy.—As is true of mania, there is also no known pathological anatomy for melancholia. It is a functional nutritional disorder of the brain, a diminished or perverted metabolism, supposed, theoretically, to rest upon a cerebral anemia, or, possibly, an autotoxemia.

Course of the Disease.—There is no such distinct prodromal stage in melancholia as in mania. The period of invasion is deliberate, and the symptoms chiefly manifested at first are gastro-intestinal disorders, dyspepsia, loss of appetite, constipation, accompanied by sensations of pressure in the head or headache, insomnia, and general malaise. The depression itself is the cardinal early psychic symptom. Melancholia, like all psychic disorders, is slow in its progress, and runs a course of from three to six months in the most favorable cases, but sometimes a year or two or three elapse before recovery takes place. Ordinarily, recovery is gradual, and is frequently accompanied by a species of reactive exaltation. Occasionally recovery is quite rapid. In women the approach of convalescence is indicated by a return of the menstrual function. In all cases improvement in physical health accompanies convalescence.

Melancholia terminates in recovery (ninety per cent.), in recovery with defect, in death, in secondary dementia, in chronic melancholia, or in a secondary paranoia.

While the majority of cases of melancholia recover completely, there are a few in which, despite apparent recovery, accurate investigation reveals a defect of the intellectual powers, a difficulty of entertaining complicated conceptions and judgments, which may easily escape the notice of the patient's friends. In a very small number of cases the mind becomes so enfeebled that the condition becomes a veritable secondary dementia, in which we discover vestiges of the antecedent melancholia in the shape of automatic phrases and movements and expressions of a depressed color, yet without any actual affective mood. The patients become negligent of person and dress in the extreme, even filthy in their habits.

A chronic persistence of the melancholic symptoms is rather more frequent as a termination than secondary dementia. In chronic melancholia we observe symptoms of either the simple depressed or the agitated form with which the disorder began, but these symptoms are diminished in intensity. The precordial distress disappears. Some of their delusions, movements, and verbal expressions become automatic, as in cases accompanied by dementia. Special forms of chronic melancholia are the insanity of negation and insanity with transformed or duplicated personality. These are very apt to develop upon a hypochondriacal basis.

A termination of melancholia in a paranoid condition (paranoia secondaria melancholica) while rare, is rather more frequent as a sequel of melancholia than of mania. In these cases there are numerous hallucinations, and a cluster of delusions, religious, persecutory, or hypochondriacal, which gradually become systematized to a greater or less degree. About half of such cases recover ultimately, the remainder passing into a condition of dementia.

Death in cases of melancholia is due to suicide, marasmus, visceral disorders, diarrhea, pneumonia, etc. A very large number of long-standing cases die of tuberculosis.

Diagnosis.—One of the most common conditions with which melancholia may be confounded is a depressed stage of general paresis. The chief points of distinction are the actual intellectual defect nearly always demonstrable in paralytic dementia, and especially the physical symptoms of paresis, pupillary changes, faciolingual tremor, characteristic speech, greatly exaggerated or lost deep reflexes, and one-sided facial weakness. The depression of the paralytic dement is superficial. His melancholy delusions are ordinarily distinguished by their inordinate and preposterous character, by the monstrosity of their contents. In addition to these points, the signs of previous syphilis and the age from thirty-five to fifty years would have some corroborative value in the diagnosis of general paresis.

A primary dementia may be misinterpreted as a stuporous form of melancholia. In primary dementia, intellectual defect is the cardinal symptom; in apathetic melancholia there is no intellectual defect, and the apathy is often clearly accompanied by painful affects from time to time.

Hallucinatory paranoia with depressive hallucinations may be confused with melancholia. In hallucinatory paranoia we have two varieties, a stuporous and an agitated form, and these have some analogy to melancholia attonita and melancholia agitata. The want of fixity and systematization of delusions in melancholia is to be remembered. The history of the patient will often reveal whether the depression is primary or not. But the differentiation is often difficult, and especially so between hallucinatory melancholia and hallucinatory paranoia. Long and careful study of the case during its progress may be requisite for an absolute diagnosis.

Senile dementia may simulate a melancholia with stupor. The age and the intellectual defect present will be in favor of the former. But senile melancholia is particularly apt to present an apparent defect of intellect.

The possibility of the melancholia being a phase of a circular insanity is also to be borne in mind.

There are instances of such a disorder as typhoid fever being temporarily mistaken for melancholia, but naturally the course of the temperature and the character of the stupor or delirium would soon correct such an error.

Prognosis.—The facts which will shape prognosis are to be drawn from what has been said previously in regard to the course and termination of melancholia. In simple forms of the disorder the prognosis is very favorable indeed, and recovery can be predicted in from three to six months. In the agitated type the outlook is less favorable, and in hallucinated and apathetic forms still less so. The catatonic variety is the least favorable of all as regards recovery.

Treatment.—The first consideration in the treatment of acute melancholia is isolation. Separation from the friends and relatives and removal from the environment in which the psychosis has developed are of the greatest importance. With familiar faces and objects about him, and with his kin offering their help and sympathies, the keenest realization of his condition is brought home to the melancholiac. He feels among them all the more deeply a sense of his incapacity, of his inability to fulfil the ordinary duties and demands of his usual daily life. Whether the patient is to be isolated by commitment to an asylum depends upon several circumstances: his means; the intensity of his malady; the presence of suicidal tendencies. There are very mild cases in which moderate travel, a sojourn in the country with a nurse, a few months at the house of some country physician or in a small private asylum, will result in recovery. But the responsibility for such a course must rest with the physician who advises it, and he must keep in mind the danger of suicide in even the mildest type of melancholia. Not a few lives have been needlessly sacrificed by the inexpertness of the consulting physician. Besides extreme watchfulness on the part of the caretaker, who is not to leave the patient alone either night or day, a modified or a complete rest-cure is to be undertaken. For mild degrees of melancholia rest in bed from 6 P. M. until noon of the next day, with plenty of out-of-door exercise during the remainder of the afternoon, is most commendable. For the more severe types, continual rest in bed is requisite. The food should naturally be easily digestible and assimilable, and the patient should be made to take considerable quantities of milk and milk products (koumiss, matzoon, somal, etc.), raw eggs, meat-juices, and stimulants, when these are indicated. Massage and general faradization (sufficiently strong to contract the muscles) are useful to take the place of exercise in cases taking the complete rest-cure. Constipation should be regularly counteracted by abdominal massage, frequent purgation, glycerin injections, enemata, etc. This is particularly necessary in cases suspected of suffering from auto-intoxication. In these cases, too, gastro-intestinal antiseptics—such as salol, gr. v, or beta-naphtol, gr. v—should be administered thrice daily two hours after eating. Ten grains of glycerophosphate of soda in a large glass of hot water a half hour before eating is also a useful remedial agent in melancholia. For sleeplessness the prolonged warm bath or the hot wet-pack is to be recommended; in the event of their failure to induce a few hours' sleep in each twenty-four hours, sleep-producing drugs are necessary. Sulphonal and trional, of each ten grains, given together at bedtime with a glass of hot milk or a cup of hot soup, are efficient in mild cases.

The opium treatment is a sort of specific for melancholia, especially when there are agitation and precordial anxiety and distress. Beginning with a medium dose three or four times a day, we gradually increase it as required. Laudanum—the solid extract—or codein may be administered by mouth. When employed hypodermatically, which is usually best, the watery extract of opium is used. It is preferable to administer morphin only in the most aggravated cases, and in these it may often be advantageously combined with hyoscin, hyoscyamin, or duboisin. It is needless to say that the opium treatment should not be made known to the patient, and it is carried out with more safety, as regards the formation of a habit, when the patient is in an institution. As the patient improves, the opium is gradually reduced until it can be finally cut off altogether. Opium does not increase constipation, except possibly for a few days when first employed; it seems actually in many cases to diminish it. Sometimes, indeed, we need to treat diarrheas that arise as a result of the opium treatment.

As soon as it becomes possible to do so, physical occupation should be begun and encouraged. A life out-of-doors, made interesting by different kinds of amusement or labor; walks, field studies in natural history (botany, ornithology, geology, physical geography, etc.), golf, bicycling, agriculture, and gardening—all of these have their place among the remedial agents at the disposition of the discerning and judicious physician.

CHAPTER VIII.

CIRCULAR INSANITY.

Synonyms.—Alternating Insanity; Insanity of double form; Insanity of double phase; Cyclic psychosis.

Definition.—Circular insanity is a form of psychosis characterized by an alternation of states of mania and melancholia. There are varieties of circular insanity which will be discussed later, but the maniomelancholic alternation is the distinguishing feature of all types of this cyclic psychosis.

Etiology.—Heredity plays an especially significant part in the causation of circular insanity (sixty per cent.). Not only do we find in the family history of the majority of these cases hereditary equivalents of different kinds, but direct inheritance of this particular variety of mental disorder is strikingly frequent.

Many degenerates exhibit a tendency to an alternating variation of mood. Sometimes they are depressed and sometimes cheerful. It is probable that this oscillation of moods in an individual with strong hereditary taint may be the rudimentary foundation upon which the superstructure of a circular insanity is subsequently laid.

Among special factors which tend to develop cases of acquired circular insanity are trauma to the head, alcoholism, hysteria, and epilepsy. The exciting causes are physical and moral, such as have been described in the chapter on General Etiology. Circular insanity is much more common in women than in men, the proportion being about four to one. Many cases develop about the age of puberty, and nearly all before the age of thirty years. The frequency of this type of psychosis as compared with other forms has not yet been determined. It is only recently that it has begun to be classified as a distinct type in our asylum statistics. Thus, the report of the Commission in Lunacy of New York State shows but ninety-six cases of circular insanity in nearly forty thousand admissions between October 1, 1888, and October 1, 1896, but the type had been recognized in the reports required from the asylums for only about a year of that time. It is difficult, therefore, to arrive at any certain conclusion, but the best authorities agree that five and perhaps more cases of alternating insanity will be found among every hundred insane patients.

Symptomatology.—The symptoms will vary at any given time according to the phase which the disorder has reached at the time of examination—the phase of depression or the phase of exaltation. The *melancholic period* may present any one of the forms of melancholia described in another chapter, from a simple depressed condition, scarcely distinguishable from the normal state of the patient, to the most pronounced melancholic syndrome. In some cases we have melancholia simplex, in others the hallucinatory variety; in some the agitation, in others stupor and catatonia. When, in any given case, the melancholic phase recurs again, it is prone to wear the same features as in the first attack. Thus, mild depression or simple melancholia, melancholia agitata, or melancholia attonita may reappear again and again as the cycle returns, with the same phase and character over and over again. While this is true in the majority of cases of circular insanity, it is not always so, for occasionally the recurring melancholia changes its type in the various sequences. As intimated in the chapter on Melancholia, there is often a species of reactive exaltation in the convalescent stage of the disease, and occasionally this reaction becomes so accentuated as to develop a maniacal condition, so that we have presented to us a picture very like that of an alternating insanity.

Like the melancholic phase, the *maniacal period* of circular insanity may vary in character from a condition of mild exhilaration and exaltation to the severest types of maniacal excitement and incoherence. As in the depressed period, there is the same tendency of the maniacal phase in its recurrences to present regularly the identical features of former attacks, though there are also exceptional instances here where subsequent outbreaks wear a different maniacal aspect.

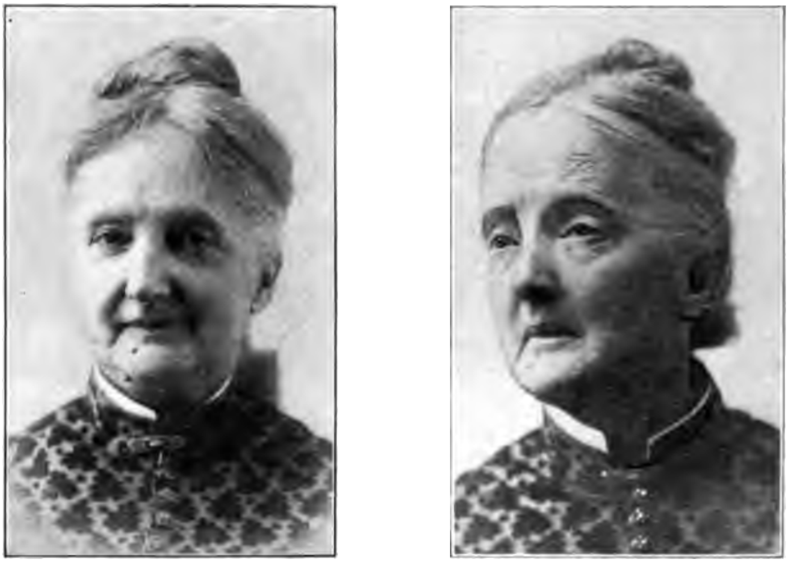

Fig. 308.—A case of circular insanity, photographed first in maniacal or exalted phase, and some months later in the melancholic phase (Dr. Atwood).

In the chapter on Mania is made mention of the fact that the convalescence from that psychosis is not infrequently characterized by a reactive depression, a lacrymose irritability. In some instances this may attain to the degree of a true melancholia, and thus place before us a cycle similar to that of an alternating insanity.

Ordinarily we recognize two degrees of intensity in circular insanity —one in which both the mania and melancholia are mild, and one in which both the mania and melancholia are severe. But there are mixed types, in which the mania may be mild and the melancholia severe, or vice versâ.

Mild types of circular insanity—instances in which both the depressed and exalted phases are so moderate in degree as not to permit of commitment to an asylum—are not infrequently met with by the practitioner, and they are often difficult cases to handle properly. Thus, I have in mind two brothers, now over fifty years of age, who are both afflicted with circular insanity, manifested in a form very distressing to the relatives. A description of one will describe the other, and not only him, but many other similar cases:

E., male, aged fifty-four, single, with hereditary taint, has for many years been subject to alternating attacks of depression and exaltation. I have seen and examined him in both phases. There is little, if any, discernible interval, but a gradual merging of one phase into the other. The depressed period lasts for from three to six months. In this, his expression is dejected; he feels that life is a failure, that he can not live long. He consults various physicians for different maladies which he thinks may account for his general malaise. He can not concentrate his mind on anything, can not read or write letters; refuses to transact the most necessary business in connection with his estate. He talks little, and broods over the mistakes and follies committed in the exalted phase of his disorder. He is rather suspicious and distrustful of his family. Sometimes he is inclined to put an end to his misery by suicide. Little by little this weight of depression begins to lighten, and he passes insensibly into a condition in which he begins to feel himself rejuvenating. Life takes on a little rosier color; his malaise vanishes, and a sense of well-being begins to infuse itself through his body. His expression changes from the fixed look of deep dejection to one of cheerful variability. In the place of quiet brooding we note an awakening interest in things about him. He begins to talk vivaciously, to be

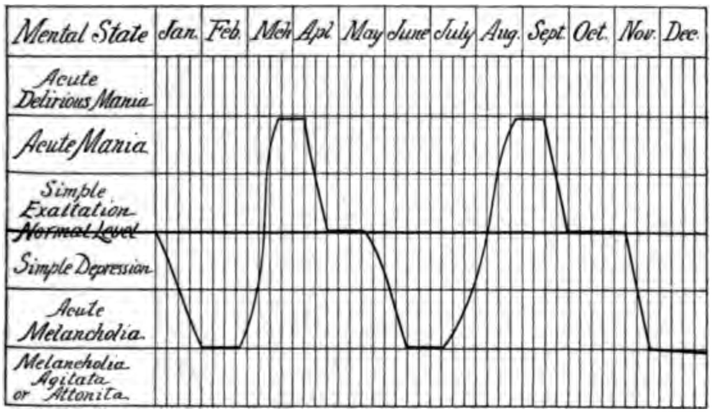

Fig. 309.—Scheme of course of disease in periodical circular insanity.

facetious and jolly, to write letters to his friends, to make frequent social calls, to take up the threads of affairs. He discards the doctors, for his health and strength were never better. He takes up some of his old hobbies, one of which is the collection of antiques, arms, plate, furniture, pictures, and specimens of ceramic art. He spends money freely, rather too lavishly. His collections are gathered together in storage warehouses, clubs, his own home, and the houses of his friends. He becomes extravagant and wasteful; enters on great schemes of money-making, in which he becomes interminably entangled and meets with financial losses. His friends expostulate, and he becomes irritable and angry. He leaves them, to live in hotels. He buys a pair of fast horses and takes a drive of several weeks all over the country for hundreds of miles around. He grows boisterous in his conversation, neglectful of the ordinary courtesies and civilities of social life, is lavish in his invitations, becomes a little excessive in drinking, is restless both night and day, travels from one city to another on the most trivial and eccentric errands. He sleeps little. Endeavors on the part of relatives to check the anarchy of his conduct bring from him threats of suits and of personal violence, and letters which are quarrelsome, offensive, even profane. With all this, there is no intellectual defect. He never has actually attempted any overt act which would put him under the control of the law, or aid in his commitment to an asylum to save the dissipation of his energies and the waste of his property. Any jury would discharge him, for his conversation would show good memory, active intelligence, keen-witted replies to all questions. Step by step this stage of exaltation begins to pass away. He sinks nearer to his normal level, resumes a more natural conduct toward his family and friends, until again the depressive elements reappear in his mental condition. Each stadium lasts for from three to six months, so that the cycle fills about one year.

Varieties.—There are two main varieties of circular insanity. One is a true circular insanity in which the phases follow each other in a perfect cycle thus: mania, melancholia, mania, melancholia, mania, melancholia, and so on. The other type is one in which there is a certain periodicity of the maniomelancholic attacks as follows: mania, melancholia, interval, mania, melancholia, interval, mania, melancholia, interval, etc. Most cases can be catalogued under one of these two headings, but there are deviations which do not exactly conform to these well-defined types, and some authors have attempted to make further, but it seems to me unnecessary, subdivisions, upon the basis of variations in the length of interval and irregularities in the sequence of the phases.

Pathological Anatomy.—Autopsies have failed to reveal any important macroscopic or microscopic changes in the brain in circular insanity. Such autopsies as I have been able to find recorded were made upon patients at an age which would naturally reveal some conditions incident to senile involution, and these morbid conditions may or may not have had relation to the mental state of the patient during life. The best that we can say, then, is that, so far as we know, there is no anatomical basis as yet discovered for circular insanity. Disordered nutrition—either insufficience or perversion of metabolism—probably underlies the manifestations of this psychosis.

Course of the Disease.—In some patients circular insanity has its inception in the melancholic period, and in others it begins with the maniacal phase. Usually the initial stadium is melancholia. The transition from the depressed to the excited phase and vice versâ is sometimes astonishingly sudden. The period of transformation may occupy but an hour or even less. In most cases the merging of one period into the other is very gradual. Another and extremely rare mode of transition is by successive alternations of depression and exaltation, an oscillating or rhythmic transformation. Still another method of change is by means of a lucid interval, brief or long, between the alternating phases, thus: mania, interval, melancholia, interval, mania, interval, melancholia, interval, etc.

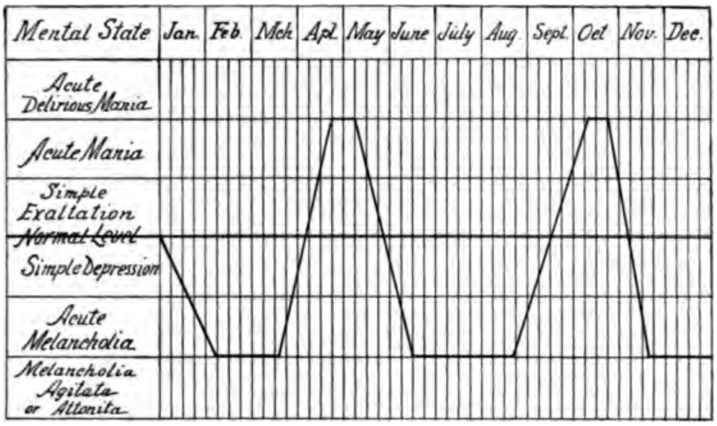

Fig. 310.—Scheme of course of disease in continuous circular insanity.

There is extreme variability in the duration of the maniomelancholic cycles. Sometimes they exhibit great irregularity of interval, from a few days to a year or more. Sometimes the maniacal phase lasts one day and the melancholic one day, so that the cycle is completed in two days. In other cases, again, the cycle is completed in two weeks, or a month, or a year. Where alternation is completed in short periods, there is a tendency to great regularity. Usually the melancholy phase lasts longer than the maniacal.

Diagnosis.—It is impossible to make a certain diagnosis of circular insanity unless at least one maniacomelancholic or melancholicomaniacal cycle has been observed. If, in a patient appearing to have an ordinary melancholia or an ordinary mania, there should be episodic oscillations of depressed and exalted emotions (as is sometimes the case in either phase of the cycle of circular insanity), then we are justified in entertaining a suspicion of alternating insanity. Even when an apparent cycle is brought to our attention in a case of insanity, it is not well to conclude too quickly that we are dealing with circular insanity, for the reactive phenomena of mania and melancholia just referred to may closely simulate the more serious disorder. The greater the intensity of the second phase in such cases, the greater the presumption of circular insanity.

There is occasionally danger of confounding a periodic mania or a periodic melancholia with circular insanity, especially if their reactive phases are notable. Naturally, if the reactive phases are very pronounced, a diagnosis of circular insanity would be justifiable.

General paralysis occasionally presents cyclical phases analogous to those of alternating insanity, but the physical symptoms, the mental enfeeblement, and the preposterous delusions of the expansive periods of paralytic dementia will ordinarily insure a correct diagnosis.

Prognosis.—Circular insanity, though made up of two of the most curable of mental disorders, is, curiously enough, itself among the most incurable. A patient seldom recovers. The disorder runs a long course over years of time, terminating ultimately in dementia.

Treatment.—All cases of circular insanity are best treated in an asylum in order to prevent suicide in the melancholic phase, and violence, excesses, and riotous extravagance in the maniacal period. Unfortunately, it is not always possible to protect the patient by this means, since juries are prone to allow every man his freedom, no matter how dangerous to himself or others, so long as he does not behave as a raving maniac before them. Even in the intervals of lucidity it is better for the patient to be under medical supervision in some institution, with the hope that the disorder may be arrested and future cycles prevented or postponed by the treatment. This treatment is based upon the principles described in the chapter on Treatment and in the chapters on Mania and Melancholia.

The rest-cure and hydrotherapy are recommended for both phases of the cycle. Hyoscin, hyoscyamin, and duboisin (gr. $\frac{1}{100}$ to gr. $\frac{1}{40}$) are useful in the excited stage, and the opium treatment in the depressed stage.

CHAPTER IX.

MANIC-DEPRESSIVE INSANITY.

MANIC-DEPRESSIVE insanity is best exemplified by cases of circular insanity in which we have recurring cycles of maniacal and melancholic outbreaks. But it is the merit of Kraepelin to have studied large numbers of patients suffering from mania or melancholia for periods of years, delving deeply into their previous histories, and following them up long after they had left his immediate professional care. This method of observation led to surprising results, not only in the matter of diagnosis, but also of prognosis. The profession had long fancied that little could be added to our clinical knowledge of the types of insanity however much remains to be learned as to their pathology. Certainly, very little brush-work was needed to complete the maniacal picture, and melancholia has been a classical syndrome since the time of Hippocrates. That these two contrasted phases of mental disorder sometimes appeared successively in the same individual, as an alternating or circular insanity, was also well known, but such combined examples were esteemed exceptional and rare. It was here that the astute German clinician stepped in and, by taking the long view over periods of years in his cases, made the discoveries which have rather upset our former classifications, and overturned some of our criteria for prognosis.

He learned by his investigations that not only is alternating insanity much more common than psychiatrists deemed it to be, but that the majority of patients suffering from what we formerly looked upon as a pure mania or a plain melancholia proved by their previous or subsequent histories to be subject to recurrences of attacks, which may be either maniacal or melancholic; and, furthermore, that there are many patients who present on careful study simultaneous manifestations of both these phases—*i. e.*, a mixture of manic and melancholic symptoms at one and the same time.

The term manic-depressive insanity is thus made to cover almost all of the old types of insanity that we formerly looked upon as separate, sharply-defined clinical syndromes, viz.: mania, melancholia, and circular insanity. And Kraepelin naturally assumes for manic-depressive insanity an identical pathology for its contrasted or mixed manifestations, though what such pathology may be is wholly a matter of speculation. Kraepelin limits the term melancholia to the depressed psychosis of senility. There is no doubt that we owe Kraepelin much for the new views thus given us. It may be, however, that modifications will be made as time goes on in this conception of the manic-depressive syndrome. The brilliant exponent of manic-depressive insanity has the advantage of us at present in having already accumulated his material requiring decades of observation for verification, while we must wait years yet with our own cases before determining for ourselves the ultimate truth of his conclusions. Many of us doubtless have cases in mind which have passed through an attack of ordinary mania or melancholia with recovery, without history of previous attacks, and with no subsequent attacks for years to the present time. We must now have the feeling that recurrence in one or the other phase is a possibility, and yet the patient may go through life without such recurrence. Under the circumstances, is it worth while to change the name of the disorder from its well-known designation to the complex phrase manic-depressive insanity? And is it altogether profitable to exchange that classical immemorial expression "melancholia" for the cumbrous phrase, "the depressed type of manic-depressive insanity," when we mean exactly what we did before, only assuming a new theory as to probable pathology? Why abandon the word "mania," when language needs condensation, for a phrase like "the manic type of manic-depressive insanity?" The clinical pictures remain the same; their interrelations are differently conceived. Some of the features of these psychoses which led Kraepelin to classify them under the one head of manic-depressive insanity, and to assume them to be equivalents of one and the same fundamental pathological process, are as follows:

The coëxistence in the same subject of alternating mania and melancholia (circular insanity).

The presence in all forms, whether excited, depressed, or mixed, of certain fundamental symptoms, such as psychical inhibition (weakened attention, retarded flow of ideas, insufficient attention, indifference) and exalted mental automatism (flight of ideas, irritability, impulses, delusions, hallucinations and illusions, fixed ideas, obsessions).

Classification of Manic-depressive Insanity.—Kraepelin divides the forms of psychoses under this heading as follows:

1. The manic type of manic-depressive insanity.
2. The depressed type of manic-depressive insanity.
3. The mixed type of manic-depressive insanity.

The manic type is separated into three forms: simple, delusional, and confusional.

The depressed type is divided into three forms: simple, delusional, and stuporous.

The mixed type is exemplified in cases of circular insanity, and also in very singular and varied mixtures of the two contrasting phases at the same time. Thus, some patients present a combination of psychomotor excitement with emotional depression, and others psychomotor inhibition with emotional exaltation. Still another group of cases is characterized by a combination of stupor with motor excitement.

A further classification of manic-depressive insanity, showing the various old forms included under this heading, is as follows:

1. Periodic insanities { Recurrent mania.
   Recurrent melancholia.
2. Alternating insanity, Circular insanity.
3. Irregular forms.

Etiology of Manic-depressive Insanity.—What has been said in previous chapters as to the etiology of insanity in general and of mania, melancholia, and circular insanity need not be repeated here. Kraepelin finds heredity a feature in about eighty per cent. of his cases of manic-depressive insanity. This psychosis forms about fifteen per cent. of all admissions to asylums. The disease generally begins before the twenty-fifth year, but a first attack may take place before the tenth year or not until after the fiftieth year of life.

Prognosis.—As described in the preceding chapters on mania, melancholia, and circular insanity, the prognosis of recovery from the existing attack of mania and melancholia is favorable, from circular insanity unfavorable. But from the new standpoint, given us by Kraepelin, we are not able to preclude a recurrence of attacks of mental disorder in a patient recovering from mania or melancholia. While the prognosis, therefore, is favorable as regards the single attack, the possibility of subsequent attacks must be carried in mind by the physician in discussing the prognosis with the family.

Treatment.—The lines of treatment for the various phases of manic-depressive insanity are sufficiently clear from the therapeutic memoranda given in the preceding three chapters.

CHAPTER X.

EPILEPTIC INSANITY.

SOME ten per cent. of all epileptics become insane. Hence the epileptic neurosis in an individual renders him about thirty times more liable to insanity than if he were normal. The psychoses to which the epileptic is subject vary extremely in character. It is my aim to give here a brief review of these. I shall not consider under this heading forms of mental disorder in which epilepsy or repeated epileptiform convulsions make their appearance in conjunction with the psychic disturbance as the result of a common cause (general paralysis, chronic alcoholism, epileptic idiocy, paralytic idiocy, etc.), but shall limit myself to the class of insanities induced by the epilepsy. It is, first of all, necessary to dwell for a moment upon some of the ordinary features of epilepsy, apart from the familiar phenomenon of muscular convulsion. The epileptic is subject to peculiar symptoms, which are looked upon as the equivalents of convulsive seizures. Among these are sudden brief losses of consciousness. The consciousness may be merely clouded or completely lost. There may be no perceptible concomitant symptoms. On the other hand, the defect of consciousness may be accompanied by some pallor of the face, a fixity of the eyes, or a partial local spasm or movement (strabismus, stammering of a few words, grimaces, lifting the arm, bowing movement of the body, turning of the head, etc.). The disorder of consciousness may be associated with an automatic dream-state, similar to somnambulism, in which complicated impulsive movements take place (automatic continuance of acts begun before the seizure, purposeless running, undressing, etc.). Vertiginous attacks may be the equivalent of convulsions. The aura of an epileptic attack may be in the form of a hallucination. A study of the psychology of epileptics in general gives us a sort of composite picture, to which all of these patients conform more or less closely. The mental attitude of the epileptic is due to a variety of circumstances. In the first place, he has a consciousness of the dreadful nature of his malady. He is in a state of expectant attention as regards the sudden blackness and prostration which are to strike him unawares at any time, in any place, like the lightning from a clear sky. He can never share the social pleasures of his fellows. The schools are not open to such as he. When he becomes old enough to work, he finds that no one wishes to employ him. Every avenue of education, every trade and calling, every road to mental progress, is barred. He is a social outcast, an object of commiseration, a burden to his friends, perhaps a family blemish to be kept concealed. The doctor is called in, and, taking, as a rule, a hopeless view of the case, abandons him to the mercy of the bromids, which further his mental, physical, and moral degradation. In this way the epileptic character is evolved. It consists of a mixture of melancholy, hypochondriasis, emotional irritability, moroseness, distrust, misanthropy, mental apathy, and dullness, often combined with morbid religious tendencies and modified by pathological psychic conditions incident to the ravages of the disease itself. These pathological mental states vary from the peculiar psychic equivalents just described to the actual psychoses of divers forms now to be detailed. Epileptic insanity is chiefly a progressive psychic deterioration terminating in dementia. But the progressive degeneration is frequently marked by episodic outbreaks of psychoses under various forms. Among these are transitory hallucinatory and stuporous disorders and chronic epileptic psychoses (under any form, such as mania, melancholia, circular insanity).

Psychic Degeneration of Epileptics.—As is well known, severe epileptic attacks are ordinarily followed by a somnolent and stuporous condition lasting from an hour or two to several days. The frequent repetition of such attacks tends to render complete recovery from such mental torpor more and more difficult. As a consequence, we observe a gradual weakening of the intellectual processes. The flow of ideas is retarded and the expression of such ideas along motor lines becomes sluggish; the speech especially has a characteristic slowness; attention is diminished and memory impaired; the concepts and judgments are built up with ever-slackening activity. In this way the epileptic may sink gradually into a deepening simple dementia. In some cases the concepts attended with ethical feelings vanish first, and to so striking an extent that acts of violence, cruelty, brutality, and crime are committed without a single inhibitory effort or a shadow of remorse. These acts often have an impulsive character.

An excessive irritability of temper is a phase of epileptic psychic degeneration. The most trivial incidents may give rise to outbursts of anger and even of overwhelming fury.

The natural hypochondriacal depression of many epileptics is frequently much exaggerated, giving rise to a sort of melancholia colored by mental enfeeblement, and by suspicion, distrust, misanthropy, and moroseness.

Occasionally, in the midst of this progressive deterioration of mind, imperative ideas and acts manifest themselves, and delirious states appear with dreadful hallucinations and delusions of persecution (paranoia-like outbreaks).

These are the marks which distinguish the psychic side of the gradually developed dementia of epileptics. The mental enfeeblement is accompanied, as in terminal dementias generally, by increase in bodily weight, hypertrophy of the subcutaneous fatty tissue, and the gradual effacement of the lines of expression in the features. We thus reach ultimately the condition of

Epileptic Dementia.—As intimated, the rate of progress of epileptic dementia is in direct proportion to the number and severity of seizures. There are cases which go on to the terminal stage without some of the peculiar manifestations of progressive epileptic degeneration just described, and others, again, in which these features are prominent. The dementia may be absolute, so that not the simplest concrete memory-picture remains in the vacant mind; the patient needs care in his person and dress, and often has to be guided and assisted in taking nourishment. His sensibilities become so diminished that he is indifferent to stimulation of any sense, and has no perception of the needs of the body as regards the bowels or bladder. He must be cared for like an infant. A persistent sexual instinct often impels him to constant masturbation.

During progress into dementia, we note the intercurrent hallucinatory states already mentioned, and the accesses of anger, with assaults and impulsive actions of various kinds. The motor memories suffer in the end to such degree that all complicated movements are forgotten. This is particularly noteworthy in the use of words, which are separated by considerable pauses. Often even the syllables are thus divided. Finally, the patient loses the power of speech altogether (aside from the actual aphasic attacks, which are not infrequently observed in connection with severe epileptic seizures).

The course of epileptic dementia is rarely rapid; it usually extends over a period of years. The cause of death is usually accident, status epilepticus, pneumonia, intestinal catarrh, inflammation of the bladder, or some other intercurrent affection. Epileptic dements exhibit a diminished resistance to diseases in general, and never attain great age.

Acute Transitory Epileptic Insanity.—The acute insanity of epileptics develops suddenly before a convulsive seizure, after the attack, or it may occur in the interval between the epileptic convulsions, commonly in the place of a convulsion, as a so-called psychic equivalent. As a rule, both onset and termination are sudden. The duration of the insanity is ordinarily from a few hours to a few days, though the attacks are sometimes shorter and sometimes longer. The symptoms are peculiar and various. The chief characteristic is the clouding of consciousness. The patient's state may be one of complete unconsciousness, though usually consciousness is not entirely lost. It is rather a condition of subconsciousness or of subliminal consciousness, with stupor. Upon this screen of clouded consciousness there is a play of multiform and bizarre psycopathic outlines—many-hued, terrible, or ecstatic hallucinations; delirium, mutism, incoherence, verbigeration, anxious states, delusions (often of a persecutory nature), or irresistible impulsions to assault, destructiveness, homicide, and suicide. Sometimes the fundamental tone of the outbreak is melancholic, more often maniacal, but the most appropriate designation of these acute epileptic psychoses is, perhaps, acute hallucinatory paranoia. There is no essential difference between them, whether the attack be preparoxysmal or postparoxysmal, or the equivalent of the paroxysm.

The stupor of epileptic insanity is distinguished from that of other psychoses by marked loss of consciousness, enfeebled attention, analgesia, sudden violence, and confusion.

We sometimes observe in connection with subconsciousness primary anxious states, resembling precordial dread, with extremely painful sensations of oppression and suffocation in the breast; and much more rarely primordial exaltation, with acceleration of the stream of ideas.

Hallucinations are mostly limited to the visual, auditory, and olfactory senses, chiefly to the first-named. The patient sees wild beasts, specters, flames, the fires of hell, wheels, gigantic threatening objects, falling walls, overwhelming waves of water; or, on the other hand, the golden gates of heaven, the jasper throne, God, and the choir of angels. He hears menacing voices, clamor and uproar, the thunder of cannon, or the singing of the hosts of heaven, the voice of God, etc. Disagreeable and noxious or pleasant odors may be perceived. A peculiarity of these hallucinations is a certain monotony of character, a general sameness, in great part due to the rather child-like constitution of the mind of epileptics. Their education and mental evolution are so often, from the nature of their malady, hampered and retarded, that they pass through life with the fancy and understanding of a child.

Incoherence of speech and lack of orientation as to surroundings are more marked in epileptic insanity than in any other psychosis.

The motor symptoms vary extremely. Sometimes we note motor inhibition attaining to complete immobility and mutism, lasting for hours, days, or weeks at a time. Such quiescence is often interrupted by sudden explosive acts of violence. Again, in other cases, we observe agitation, restless wandering about, purposeless and impetuous running hither and thither, assaults, destructiveness, and, rarely, complicated acts, like theft and other petty crimes. A condition of religious ecstasy is not uncommon. The patient may feel himself wafted to heaven, where he converses with God, Christ, and the disciples.

In some rare instances epileptics are subject to dream-like states of subconsciousness, similar to somnambulism, in which complicated acts are carried out. Like the somnambulist, such patients may seem to be conscious, may comport themselves in speech and conduct in a perfectly natural manner, and in this condition, which may last for hours, days, or even weeks, commit offenses against the law, wander off as tramps, or do some extraordinary thing in following the imperative, childish, silly, or fantastic ideas which control their dream-state.

The disorders of memory incident to transitory epileptic insanity are both interesting and important. There may be, upon recovery, absolute amnesia as regards everything that has taken place. There may be remembrance of much that has occurred immediately after the insanity has passed, with subsequent amnesia. There may be complete amnesia at first, with glimpses of remembrance afterward. There is rarely any persistent recollection of the events of the psycopathic state.

As has been stated, the rule is for these transitory epileptic insanities to exhibit a sudden onset and a sudden termination. The longer the duration, the less abrupt the cessation. The majority of these patients recover, but recurrence is, of course, frequent. Termination in a chronic condition is rare. Occasionally, death takes place from exhaustion, intercurrent maladies, or from a convulsive seizure or series of attacks during the psychosis. Recurrences tend to hasten a psychic degeneration ending in dementia.

The epileptic nature of such insanity as is here described, where the history is not known, is determined by the following characteristics: (1) Sudden onset and abrupt termination; (2) the terrifying or ecstatic nature of the hallucinations and delusions; (3) disturbance of consciousness and stuporous condition; (4) impulsive acts; (5) dream-states; (6) amnesia.

Chronic Epileptic Insanity.—Aside from epileptic dementia, the acute epileptic psychosis just described may take a chronic course, or assume a periodic form, with little improvement in the intervals between the exacerbations. There are cases which closely resemble chronic mania in their long course, and others in which melancholia is the predominating feature. The epileptic attacks to which these patients are subject are naturally the distinguishing feature, and a special color is given such cases by the epileptic psychic degeneration. Occasionally a true circular insanity is presented, with its alternating maniacal and melancholic phases.

Treatment.—Most cases of pronounced epileptic insanity require commitment to an asylum. Their proclivity to sudden accesses of rage and fury and to impulsive acts of violence necessitates this course. Where there is simply a moderate amount of psychic degeneration this course is not necessary.

The treatment should be, in the first instance, prophylactic; but, after the development of the psychosis, it consists of a combination of the treatment of ordinary epilepsy with that of the particular type of insanity presented.

Preventive therapy is concerned with the counteraction of the many elements which favor mental deterioration, with the mitigation of the epileptic's early sufferings, with the reconstruction of his environment. It may be called the moral and manual method. The moral part of it is the opportunity for education, regular occupation, and recreation. The manual and hygienic part of it, the acquisition of out-of-door trades or callings—muscular exercise, which in itself serves to reduce the number and intensity of convulsive seizures. I may be pardoned for dwelling somewhat longer on this subject of preventive therapy, and for allowing my pen to go over the same lines which it has traveled so often in past years, because I am convinced that this moral treatment marks the greatest stride in advance made for centuries in the therapeutics of epilepsy. For ages drugs have been exploited as helpful or curative; but, after all, little has been accomplished from the standpoint of materia medica. Only of late years has the moral treatment become prominent. As a rule, the epileptic patient was dismissed by his physician with a prescription of uncertain value and possibly a few general directions as to diet. It was not known to the practitioner—or, at least, he did not concern himself about the matter—that the epileptic could gain admission to no hospital of any kind; that he had no associates, occupation, or recreation; that, debarred from the schools, he grew up uneducated, and with a tendency toward retrogression rather than progress; and that, without teaching, reared in idleness, suffering from a dreadful malady, neglected in body and mind, he could find shelter at last only in the almshouses and insane asylums, these being the only institutions open to him. Yet, in by far the majority of cases of epilepsy, the attacks rob them for but brief intervals of the capacities for study, work, recreation, and social pastimes, which they possess in common with their more fortunate fellow-men. Hence the adoption of a scheme of colonization of epileptic dependents on the model of the great German colony at Bielefeld, of which the Craig Colony, in the State of New York, is an example. The Craig Colony consists of a tract of nearly nineteen hundred acres of land in the most fertile, productive, and picturesque valley of the State (the Genesee Valley). Upon this are already some sixty to eighty buildings, with accommodations at present for but 840 patients. Over eleven hundred epileptics are now on the list of patients awaiting admission. Here they are to be given an education in the various branches of learning taught in the public schools, to be instructed in every kind of industry, to be treated each and every one for epilepsy, and to be offered a home in a sort of village life, where they will no longer have the feeling of social ostracism, or be debarred from the privileges of intellectual and moral development enjoyed by the rest of mankind.

The out-of-door life in a farming community has already had wonderful results, which may be learned from the annual reports of the colony. It will suffice to say here that the average reduction in frequency of attacks among all the patients has been fully fifty per cent., and that the mental and moral regeneration of the beneficiaries has been truly remarkable. What the effect of such change of environment must be as a prophylactic against psychic degeneration and insanity can not be estimated. We may now briefly touch upon the medicinal and surgical treatment of epilepsy. The old drugs—borax, nitrate of silver, belladonna, and the bromids—have their uses. One is valuable in one case and not in the other; and each patient, where the disease is idiopathic, and no etiological indication exists for the preferment of an especial agent, must be experimented upon with one drug after another for two or three months at a time, until a satisfactory remedy is discovered. Upon the whole, the bromids are most effective as a general antispasmodic for all cases. While the bromids are, perhaps, the most useful remedy we can employ as an antispasmodic in many cases of epilepsy, their exhibition in every case is not advisable. With a considerable number of patients the bromids are entirely ineffectual; with no small number, too, very serious symptoms, such as acute bromism, increase of seizures, and even insanity, supervene upon their use. In many of the cases where actual good is done by the bromids in reducing the frequency and severity of the attacks, the concomitant symptoms are such that it becomes questionable whether the remedy be not, after all, worse than the disease. The writer makes it a practice, therefore, to exhibit the bromids with caution, and never to employ them until the series of less harmful, but often quite as efficacious, remedies for epilepsy have been tried in vain.

There are some new drugs and remedial methods that have come into vogue of late which are worthy of attention. In the first place, there is simulo, a South American plant of the hyssop family, the tincture of which is given in doses of one to two or three drams three times daily. After an experience in many cases for several years, I would say of simulo that it deserves trial in most cases; that it is perfectly harmless, which can not be said of the bromids, borax, belladonna, and some other drugs; that in a few cases it has been extremely beneficial in my hands, and that in most cases it has no effect at all. Simulo combined with small doses of bromid acts very well. The so-called opium-bromid treatment of Flechsig is of value for many patients, especially in old and obstinate cases where all other agents have proved ineffectual. This treatment consists of the administration of opium for some six weeks, beginning with one-half to one grain three times daily, and increasing gradually until ten to fifteen grains a day are taken, when the use of opium is suddenly stopped, and bromids in large and gradually reduced doses are given (thirty grains four times daily, to begin with). I had used in certain cases of epilepsy for some years codein with considerable success, but this combination of the opiate with bromids is still more satisfactory.

Adonis vernalis conjoined with the bromids, as recently suggested by Bechterew, is an efficient method of treatment, from which, in several instances, I have had gratifying results. Digitalis, which has properties similar to Adonis vernalis, was formerly frequently given in epilepsy, but the new combination seems to be much more efficacious.

There are a few cases of epilepsy in which careful investigation indicates self-intoxication as a factor. In these an excess of ethereal sulphates (indican) in the urine, together with periodical or constant attacks of gaseous diarrhea, are almost positive manifestations of putrefactive or fermentative changes taking place in the alimentary tract. It is remarkable how much benefit may be obtained in such patients by the regulation of the diet (milk and its modifications, koumiss, matzoon, somal, rare or raw beef, eggs, green vegetables, and special breadstuffs, like Zweiback, Huntley & Palmer's breakfast biscuits, and Voebt's *biscotte de legumine*), by the frequent drinking of hot water and the occasional flushing out of the large intestine by hot water, and by the use of certain intestinal antiseptics, given two hours after eating, with plenty of water (beta-naphtol or salol, gr. v).

The remarkable effect of the thyroid extract upon general nutrition would naturally suggest the advisability of its administration for experimental purposes in some of the nervous diseases which we are accustomed to look upon as due to nutritional disturbances in the nervous system. With this idea in view, I have employed it in a good many y, in a number with very good effect. Especially note- y was mental improvement in several cases of epilepsy with ntly considerable dementia. It is worthy of more extended trial.

le from the remedies for the epilepsy just described, we need ially to employ certain other drugs for particular conditions, s status epilepticus, maniacal outbreaks, pronounced melan- states of terror, etc. In status epilepticus rectal injections of , gr. xx, with an ounce of starch-water, repeated at intervals of three hours if needed, give the most satisfaction. In great ideo- excitement we should use hyoscin, hyoscyamin, or duboisin ermatically, in doses of $\frac{1}{100}$ to $\frac{1}{40}$ of a grain. In anxious melan- conditions morphin hypodermatically is, perhaps, the best allevi- agent to exhibit.

ie question of trephining must naturally come up in certain cases leptic psychoses where trauma to the head is evidently the cause epilepsy and psychic degeneration. The following points are to ken into consideration as a guide in this matter:

In the very small number of cases having injury to the head as ise the epileptic habit is so strong, and the changes in the brain usually so old and deep-seated, that an operation, as a rule, does cure, and seldom permanently diminishes the frequency of the ks.

Of miscellaneous traumatic cases, where a surgical procedure justifiable and is undertaken, a cure of the epilepsy may be ably expected in, perhaps, four out of every hundred cases ed upon.

3. The removal of a cicatrix from the cortex, supposed to be the epileptogenic nidus, will naturally be followed by the formation of a new cicatrix in the surgical wound—the creation, therefore, of a new epileptogenic center.

4. The more recent the injury, the greater will be the promise of lasting benefit.

5. In cases of traumatic epilepsy with marked epileptic psychoses (recurrent attacks of rage, fury, violence, destructiveness, etc.) trephining would be justifiable as a possible means of diminishing the severity, danger, and frequency of the maniacal attacks, even though the epilepsy itself or the psychic degeneration might not be improved.

CHAPTER XI.

DEMENTIA.

Secondary; Senile; Primary.

Definition.—"Dementia" is a term employed to designate simply a general enfeeblement of all the mental faculties. It is often used improperly by the laity as synonymous with insanity. But in medicine it signifies only a general weakening of a mind once normal. Hence it is not applied to congenital mental weakness. The term "idiocy," with its various degrees, includes all of these congenital psychic defects. There are innumerable gradations comprised in dementia, from the merest dullness to profound deficiency or complete loss of all the intellectual faculties. Such enfeeblement of the mind may be the result of serious cerebral diseases or disorders, such as epilepsy, alcoholism, syphilis, etc., when the dementia is qualified as epileptic, alcoholic, syphilitic dementia, etc. It is often a sequel to acute insanities, like mania and melancholia, and to chronic psychoses, like circular insanity and paranoia, and hence the distinctive term *secondary dementia* applied to such examples. It takes the chief part in the syndrome of paresis, so that that disorder is often entitled "paralytic dementia." Progressive mental enfeeblement not infrequently accompanies senile involution and organic changes in the brain incident to that epoch of life; hence the well-known disorder called *senile dementia.* Finally, there is a form of mental disease characterized in the main from the very beginning by extraordinary psychic enfeeblement, and this malady is classified as an *acute* or *primary dementia.*

Under the heading of dementia we shall now consider separately the more important forms of dementia just described—viz., secondary dementia, senile dementia, and primary dementia.

SECONDARY DEMENTIA.

Secondary dements make up by far the greater number of the patients accumulated in our large asylums. Every year the number is augmented by the increment of new cases which enter upon this terminal and incurable condition. It has been estimated that some two-thirds of the patients in asylums belong to this category.

Symptomatology.—The cardinal symptoms are defect of memory, deficient ideation, and feebleness of judgment. There is no longer any logical coördination in the flow of thought. The speech is incoherent when there are any ideas at all to seek expression. Some patients chatter a great deal with no coherence or meaning, the only connection of one word or phrase with another being similarity of sound. Other patients are absolutely silent. Hallucinations are often present, more particularly at the period of transition from the antecedent psychosis to the terminal dementia. Delusions may also exist, but they are vestiges of the delusions of the primary insanity carried over into the secondary condition. The feebleness of mind is shown especially in the state of the emotions, which have a child-like simplicity of expression. These patients laugh boisterously over nothing, weep about trifles, and are easily enraged without sufficient motive. Naturally, all of the higher concepts are lost, especially those of esthetic and ethical character. The habits become depraved and loathsome in extreme cases. Masturbation, destructiveness of clothing, besmearing of the person with and eating of filth are frequent manifestations in the lowest degrees of dementia. The patients become robust and fat. They lose all expression, save some single, automatic, fatuous smile, angry frown, furtive look, or aspect of misery, which may linger as a legacy from the previous psychosis. They swallow anything they can get hold of; they collect pebbles, pieces of paper, string, glass,—in fact, all sorts of rubbish,—which they either pocket or use for personal ornament. Many show a proclivity to automatic movements, analogous to those observed in idiocy, such as anteroposterior or lateral oscillations of the body. Occasionally these movements are more complicated, taking the form of grimaces ; gesticulations with the fingers, hands, and arms ; running to and fro, running in a circle, whirling round on the heel, etc. A considerable loss of sensibility to pain is generally noticeable in secondary dements. Hematoma auris is common among them.

It is customary to classify secondary dementia into two groups, bespeaking contrasting syndromes—viz., *apathetic* and *agitated* dementia.

Patients with apathetic dementia are expressionless, never speak, crouch or lie about the floor or in corners in the most negligent attitudes, and cover their heads with their clothing.

Patients with agitated dementia are the restless ones just alluded to, and such cases as show a tendency to accesses of excitement. These outbreaks are doubtless aroused by processes going on within the organism, since they occur without any external exciting cause.

Secondary dementia may be regarded as a presentment of the mind in ruins. The storm has swept by with its havoc and devastation. After its fury has been spent, a certain amount of placidity remains. In the midst of this calm we note the wreck that has been wrought. Some of the old architectural details stand out, so that we may still recognize what manner of mind it was. There are residua, too, of the destructive agent that was at work, traces that indicate the character of the brain-storm when it was at its height.

The foregoing are the main outlines of secondary dementia, but, as intimated before, there are innumerable gradations of mental enfeeblement in these cases. A large part of the work done in and about asylums is performed by secondary dements in whom the intellectual decay is not extreme.

Course and Prognosis.—The course of secondary dementia is chronic. Usually, there is no progressive increase of mental enfeeblement, rather a pause after a time, when the mind reaches a certain plane of deterioration. Here the process becomes stationary. The patient leads his mindless, vegetative existence for years and years, sometimes to a good old age, because in the asylum he lives a life of perfect regularity as to food, sleep, and exercise, and is snugly protected from the vicissitudes of weather and of the daily struggle in the outer world.

These patients never recover. Actual tissue-alterations were made by the psychosis which swept through their brains.

Pathological Anatomy.—Thickening of the vascular walls, distention of the perivascular spaces, destruction of ganglion-cells and cortical association fibers, and some narrowing of the cortex—these are the main postmortem findings.

SENILE DEMENTIA.

This is a progressive mental enfeeblement at the period of senile involution, dependent upon organic changes in the brain : therefore, a chronic organic psychosis.

Etiology.—Heredity has been noted in some fifty per cent. of the cases. Males and females suffer about equally. The disorder rarely appears before the sixtieth year. Mental stress and physical illness, together with the senile involution, are the chief etiological factors. In most of the cases arteriosclerosis takes part in the causation of the disease, inducing, as it does, general malnutrition of the brain, as well as frequent local degenerations of small or large extent.

Symptomatology.—The earliest symptom is failure of memory. The most recent memories disappear first in a sort of chronological order. After a time the patient fails to recognize any of his surroundings or any of the people about him. He converses with those near him, and miscalls them, as if they were old friends of long years ago. He lives over old events as if they were now enacted. Later on even these old memories vanish also. With failing memory, the judgment-associations perish. The patient commits many breaches of decorum, and later, with the degeneration of ethical feelings and the ascendancy of coarser instincts, may become very negligent, indecent, and unclean in habits; may pilfer and destroy things; may expose his person, masturbate, or attempt liberties with little girls, etc. His loss of judgment may induce him to foolishly squander his money and properties.

Illusions and hallucinations begin to manifest themselves. They are usually of terrifying character.

Delusions make their appearance. These are nearly always persecutory in nature, and arise either as primary ideas or as the result of depression or on the basis of hallucinations. Next to delusions of persecution in frequency, we observe hypochondriacal delusions, with contents modified by the weak-mindedness present. Delusions of approaching poverty are quite common.

The underlying mood is often melancholic; an exalted mood is extremely rare. Changeability with irritability is perhaps the most usual affective condition.

The behavior of these patients in relation to night is noteworthy. Illusions, hallucinations, delusions, and emotional states all become more pronounced at night. A striking feature, too, is extreme motor restlessness, especially at night. These patients try to get up from bed, to wander about the house, to get away from something or somebody. Sometimes true melancholic anxious states come on and lead to attempts at suicide.

So far as bodily symptoms are concerned, we note foremost among them a general senile decrepitude, to which are added senile tremor of the hands, and often various stigmata of focal lesions in the brain (aphasic and paraphasic attacks); sometimes hemiparesis, monoplegia, hemiplegia, etc., complicate the picture. The patients often complain of severe pains all over the body, of vertigo, ringing in the ears, sparks before the eyes, etc. Often, too, there is noticeable diminution of sensibility to touch and pain in various areas, or over the whole body. Occasionally an especial color is given to the symptoms described by true maniacal or melancholic phases appearing in the course of the disease.

Course and Prognosis.—Senile dementia develops gradually upon the basis of senile psychic degeneration, and lasts, ordinarily, from three to five years, sometimes with remissions which are never so noteworthy as the remissions of paralytic dementia. In rare instances an acute course is taken, the disease terminating by death in a few months. Paralytic attacks are not infrequently observed in the course of the malady, giving it a certain analogy to paresis. The prognosis is unfavorable, as the disorder is incurable and progressive to a fatal end.

Diagnosis.—The most important indications for diagnosis are defects of memory and judgment and acts dependent upon loss of ethical feeling.

Pathological Anatomy.—We observe at autopsy chiefly the following conditions:

1. Osteophytic deposits on the inner surface of the skull.
2. Pachymeningitis hæmorrhagica interna (more frequently even than in paralytic dementia).
3. Opaque and thickened leptomeninges.
4. Increased fluid, subdural, and in the meshes of the pia-arachnoid.
5. Distention of the ventricles with serum, and granular ependyma.
6. Extreme narrowing of the cortex, with gaping sulci.
7. General endarteritis deformans (often with foci of softening and hemorrhage).
8. Wide-spread degeneration of ganglion-cells and association fibers.

Treatment.—Many cases of senile dementia can be treated at home. It is only when tendencies to suicide, sexual immoralities, waste of property, and great ideomotor excitement are exhibited that commitment is necessary. The bromids are the best hypnotic for these cases. Paraldehyd is extremely useful, too, since it is efficient as a hypnotic and does not injure the circulation or affect the digestive apparatus. In melancholic phases opium acts well. Hyoscin and its congeners are not to be recommended because of their depressing action on the heart.

PRIMARY DEMENTIA.

Synonyms.—Acute dementia ; Acute curable dementia ; Stupiditas.

Definition.—Primary dementia is an acute curable psychosis characterized by ideomotor inhibition and apathy. The inhibition of thought may attain to the degree of complete cessation of the psychic functions, and that of motion to complete immobility.

Etiology.—This is essentially a disorder of youth. A rare disease in itself, it is chiefly encountered in young persons between the ages of puberty and thirty years. After thirty-five it is extremely infrequent. A neuropathic constitution is found in some sixty per cent. of the cases. Any mental or physical stress that induces exhaustion of the nervous system may act as an exciting cause of primary dementia. Fright, concussion of the brain from trauma, hemorrhages, frequent child-bearing, physical and mental overwork or overexertion, and masturbation have all been cited as etiological factors.

Symptomatology.—The development of the malady is gradual. At first there is difficult concentration of the thoughts with loss of interest in everything and a certain restlessness. The patient perceives a lack of energy in his idea-associations ; nothing suggests thoughts to him, and he begins to feel a sort of depressed wonder at his own condition. Complicated processes of thought become impossible, and even the simplest concrete memory-pictures are difficult of recollection. He can not recall the countenances of his friends, the position of the furniture in his room, the situation of his home, the events of the past or of yesterday. He feels his head empty of ideas. Things seem to grow distant ; voices sound far away. The senses become blunted and respond at first slowly, later not at all, to stimuli. The patient sinks deeper and deeper into a dream-state. His face becomes expressionless, his eyes staring into vacancy. He makes no response to questions. He pays no attention to his surroundings, to his dress, to his physical needs. He grows anesthetic and analgesic. The cutaneous reflexes are markedly diminished. The pupils are widely dilated, and react but sluggishly. The tendon-reflexes are exaggerated. There are no delusions, hallucinations, or illusions, as a rule, though in some rare instances there may be some transient manifestation of such symptoms. The immobility is flaccid in character, only seldom presenting any indication of spastic tension. For hours and days he will stand, sit, or lie in one place. He is usually speechless, but if an attempt is made to utter an interjection or phrase, the voice is so low as to be little more than the movement of the muscles of articulation. The pulse is small and weak, the heart-action retarded, the temperature subnormal, the respiration shallow.

A peculiar feature of the condition is the occurrence of sudden episodic periods of excitement, with a certain amount of exaltation lasting

or two, in which the patient runs about, sings, dances, and talks
tly.

are forms of primary dementia which are more or less compli-
melancholia, stuporous paranoia, and neurasthenia.

and Prognosis.—The psychosis lasts from a few months to a
ore, and about three in five gradually recover. Most of those
ver show a defect of memory for what has occurred. Some
cover incompletely, and some undergo an imperceptible tran-
nto secondary dementia.

nosis.—The chief difficulty in diagnosis lies in the differentia-
apathetic forms of melancholia from primary dementia. From
pression, attitude, gestures, and speech, one determines the exist-
f the anxious state or hallucinations which lie at the base of
holia passiva or melancholia attonita. The history of the
will distinguish congenital or acquired idiocy from this form
ity.

ological Anatomy.—No physical basis has been established
disease. It is regarded as a purely functional psychosis.

nent.—Mild cases may be treated at home under propitious
At the same time, most of these patients are better off in
, where the discipline, regular life, and expert care favor speedy
Rest in bed and overfeeding are requisite at first. Regular
erapeutic measures are of value (at first short warm baths, later
vers and spinal douche). Medicines are of no especial value
n the episodic periods of excitement, when the bromids may be
employed, together with hot wet-packs.

CHAPTER XII.

DEMENTIA PRAECOX.

Dementia præcox is a disease beginning usually in early life, and characterized chiefly by a more or less marked and peculiar enfeeblement of the mind, but manifesting upon this basis a considerable variety of symptoms, such as emotional indifference, weakness of judgment, flightiness, verbigeration, automatic obedience, catalepsy, echopraxis, stereotypy, negativism, mutism, impulsive actions, affectations, grimaces, and unemotional laughter, delusions of a depressed or grandiose nature, and hallucinations.

It is not easy to offer a brief and clear definition of dementia præcox and I have made the above from an analysis of Kraepelin's descriptions of the multiform phases of this psychosis. He has brought together under this name a group of mental disorders, the distinguishing feature in all of which is a special type of dementia most clearly outlined in terminal conditions.

There are cases in which all of the psychical functions are equally enfeebled, as in the disorder which we have been accustomed to call primary dementia, and which type is now included in the new category. These are exceptional. The characteristic of the enfeeblement of mind in dementia præcox is the inequality of weakening of the several faculties, a sort of selective deterioration.

Kraepelin includes in this large group of cases our old class of cases known as primary dementia, the catatonia of Kahlbaum, the hebephrenia of Kahlbaum and Hecker, and the large group of cases that we in this country have been in the habit of calling chronic mania and chronic melancholia according to the nature of the delusions presented (grandiose or persecutory) and which are held by Kraepelin to be paranoid types of dementia præcox. True paranoia, or chronic delusional insanity, is not included.

Classification.—This author, therefore, divides his group dementia præcox into three types of the disease, viz.:

1. The hebephrenic type.
2. The catatonic type.
3. The paranoid type.

It must be borne in mind that these types are more or less interrelated and often run into each other.

Etiology.—Dementia præcox, according to Kraepelin, represents some fourteen to fifteen per cent. of all admissions to asylums, of which five to six per cent. would represent the hebephrenic and catatonic types and nine to ten per cent. the paranoid group. This disorder is essentially a disease of early life. In a study of 296 cases Kraepelin found that sixty per cent. began before the twenty-fifth year, though typical examples may be noted at any age. In the earlier years the disease is more apt to appear as a simple, gradually progressive dementia (hebephrenic type), a little later in adolescence as an acute or subacute form with catatonic symptoms, and still later as a paranoid condition. The two sexes are equally liable to the disorder, though there are more males affected by the hebephrenic form and more females by the catatonic and paranoid forms. Heredity was noted in seventy per cent. of the cases, and physical stigmata of degeneration are frequently to be observed. In ten to eleven per cent. of Kraepelin's cases the development of the disease was preceded by severe acute diseases, such as typhoid fever and scarlet fever, but as usually years had elapsed between the two episodes little value is to be attached to such relations. Alcoholism seems to have no place as a cause. It is noteworthy that more than six per cent. of Kraepelin's cases developed in prison.

As to the pathological basis for dementia præcox, Kraepelin is of the opinion that we have to deal with an actual chemical injury to the cortical cells causing their deterioration or destruction, and that the origin and development of the psychosis are best explained by the theory of an autointoxication arising possibly in connection with processes going on in the sexual organs. He is led to the last idea by the unusually close association of the disease with the age of development, with menstrual disorders, and with pregnancy and childbirth.

Another view which has perhaps wide credence is that the original impetus to psychical development implanted in an individual becomes prematurely exhausted, and the intellectual powers give out, dwindle, and disappear.

Symptomatology.—Let us first take a view of the general symptoms of dementia præcox and later seek a mental grasp of the syndromes presented by the three types mentioned above. The underlying characteristic of *psychical enfeeblement* common to all has already been dwelt upon.

The simple *perception of external impressions* is little, if at all, impaired. The patient appreciates all that goes on about him far more than one would suppose from his behavior. One is surprised to learn that patients apparently wholly stupid and dull will take note of innumerable remarks and events in their neighborhood, suddenly revealing their lucidity by some apt observation.

In consequence of this, *orientation* is seldom disturbed in dementia præcox. As a rule, the patient knows where he is, recognizes those about him, and clearly appreciates time.

Hallucinations and *illusions* are common, especially in acute or subacute conditions. The auditory are most frequent, after which come hallucinations and illusions of sight and common sensation (feelings of currents passing through the body, of being touched, or of influences). These sensory symptoms tend at first to be of unpleasant nature and to distress and depress the patient, but are later received with unconcern, or at times with amusement as if they were a species of theatrical performance.

Consciousness is in many cases clear throughout, though occasionally somewhat clouded in excited or stuporous states. On the other hand, the faculty of *attention* is regularly subject to serious disturbance.

One of the most prominent symptoms is the *failure of interest.* Though the patient perceives what takes place in his environment, he contemplates all with indifference.

Memory is relatively little impaired in dementia præcox, except after the lapse of years, when some reduction in the faculty may be noted.

There is a *retardation in the flow of thought,* so that ideas are associated with difficulty even to the extent of an incoherence in which no possible connecting link can be perceived. But it must be conceded that this incoherence is often more apparent than real. It is very common to observe stereotypy of language, the senseless reiteration of phrases for days, weeks, months, playing upon words, rhyming, and other forms of *verbigeration.*

The faculty of *judgment* is affected in all cases. Transitory or lasting *delusions* develop in a very large number of cases. In the early stage of the malady the delusions are of depressed character, hypochondriacal, melancholy, persecutory, but later on, grandiose ideas appear, either in association with the depressed ideas or displacing them. Such delusions, as a rule, are transitory, changeable, silly, and senseless, apparently because of the rapid advance of intellectual weakness. In the paranoid type they are less transitory and variable, but even here grow more and more disconnected and less prominent as the disorder progresses.

There are, as a rule, profound and striking disorders in the sphere of the *emotions.* An anxious or sad state marks the beginning of the disease in an extraordinarily large number of cases. Rarely there is mirthfulness with constant laughing at this early stage. But more important than these transitory conditions of depression or exaltation is the invariable development in this disorder of *emotional deterioration.* This, indeed, is one of the fundamental characteristics of dementia præcox. The failure of interest already alluded to depends in a measure upon the general emotional decadence, since the faculty of attention has an emotional foundation. The patient feels neither sadness nor joy. He is indifferent to his relatives and friends, to his old occupations and pleasures, to pain, to fears, to desires. He is insensitive to injuries, to bodily discomfort, to uncomfortable physical positions, to pricking with a needle, but not always to hunger. The presence of food will often arouse an interest that is otherwise wholly lacking.

This emotional indifference, together with deterioration of will-power, gives rise to numerous morbid manifestations in the domain of *conduct.* The patient is often practically will-less and has lost every ordinary incentive to normal behavior. He sits stupidly about, negligent of person and dress and of the demands of nature, or at times develops some sudden imperative motor activity which may reach the degree of a stormy maniacal excitement. Impulsive actions, silly and senseless, often dangerous and destructive, are manifested, but wholly without will or purpose. They are the planless expression of an inner tension. Very often the automatic impulse will be reiterated over and over again in either language or conduct, constituting *stereotypy*. The patient will repeat the same words or phrases over and over again, or turn backward and forward or round and round, or move the hands to and fro, or take attitudes for hours. A very singular symptom in this connection is *negativism*. Every incentive to some sort of motor expression is met and overcome by a counter-incentive, by an inhibition. The will is blocked, as Kraepelin describes it (*Willensperrung*). To the category of negativism belong mutism; resistance to feeding, dressing, undressing; obstinate resistance to all that physician or nurse tries to do for the patient; the holding in of the saliva and excretions; the creeping under the bedclothes; the turning away when spoken to; the inaccessibility to all requests and influences. Negativism varies in extent and degree in various patients and often in the same patient. It is not uncommon for a condition of negativism, quite unsusceptible to outward influences, to be broken through by some sudden senseless motor impulse arising in the patient himself. After the impulse has been executed the patient relapses as suddenly into the former resisting state.

With such reduction and perversion of the will in dementia præcox it is not surprising to observe at times in every case some degree of *suggestive automatism*, such as echolalia, echopraxia, and catalepsy.

The *physical disorders* of dementia præcox have no pathognomonic value, though many have been studied and described. Among them are attacks of syncope; epileptiform, apoplectiform, and hysterical seizures; localized spasms; tetany; transitory paralyses; aphonia; singultus; choreiform movements; "athetoid ataxia"; aphasia; exaggerated reflexes and increased mechanical irritability of the muscles; dilated pupils usually; vasomotor disorders (cyanosis, œdema, dermographia, hyperidrosis); increased salivary secretion; pulse slow or rapid, often weak and irregular; subnormal temperature; amenorrhea; diffuse enlargement of the thyroid gland; anæmia; emaciation in acute and subacute conditions, but rapid increase in weight in later stages.

Having thus passed over cursorily the general symptomatology of dementia præcox, it remains to place before the student rather briefly the main features of the three types described by Kraepelin. The *hebephrenic form* is characterized chiefly by a simple, progressive enfeeblement of the mind, varying in degree, and manifesting itself usually as a subacute, sometimes as an acute psychosis.

The *catatonic form* is distinguished by peculiar conditions of stupor or excitement, with symptoms of negativism, stereotypy, and suggestibility, and tending mostly to termination in dementia. When Kahlbaum first described catatonia he included under that head cases of melancholia attonita, stupor, acute dementia, etc.

The *paranoid type* is characterized by a rapidly developing mental weakness with preservation of lucidity, and by a chronic delusional and hallucinatory state.

The author separates this type into two main groups: one, dementia paranoides (with permanence of countless disconnected, constantly changing persecutory and grandiose ideas, associated with moderate excitement), and a second group characterized by singularly fantastic delusions, innumerable hallucinations and illusions, a more connected development, lasting a few years and then disappearing or terminating in confusion.

Prognosis.—The prognosis is unfavorable, most cases ending in permanent dementia; but a few do recover. It is to be noted that remissions, similar to those of general paresis, are frequent in this psychosis; hence patients may seem to have recovered fully and yet in a few days, months, or years there is a permanent return of the malady.

Treatment.—There is nothing specific to be said in regard to treatment, and the physician must be guided by the general measures described in other chapters of this book. In the early stages there is a possibility of arrest of the disorder by paying great attention to everything bearing upon nutrition (out-door life, exercise, overfeeding, tonics, etc.), by effecting a change of environment, and sometimes by suggestive therapeutics.

CHAPTER XIII.

PARALYTIC DEMENTIA.

Synonyms.—Dementia paralytica; Progressive general paralysis; General paresis; General paralysis of the insane.

Definition.—Paralytic dementia, as its name implies, is a disorder characterized chiefly by progressive enfeeblement of the mind, together with a progressive general paralysis of the whole body. It is essentially a cortical disease, but its symptomatology is frequently modified by spinal complications. The psychic symptoms, in addition to the characteristic progressive dementia, present multiform phases, neurasthenic, hysterical, hypochondriacal, melancholic, maniacal, circular, paranoiac, etc. An expansive phase with delusions of grandeur is very common at one period or another in the course of the malady.

Etiology.—Intellectual overwork or strain, working on a foundation impaired by syphilis or alcoholism, or both, may be said to be the chief cause of general paresis. Heredity, undoubtedly, plays a part in the causation of this form of mental disorder, though perhaps not so great as in other classes of insanity. The rôle of heredity has been variously computed at from ten to forty per cent. As regards sex, it may be stated that on an average, among all classes of society, twelve times as many males as females are affected—the disproportion seems to be less among lower orders of people. The age of onset is usually during the fourth or fifth decad, bespeaking in general the climacteric period of human life. But general paralysis may be encountered at almost any age. Nearly one hundred cases have been recorded as occurring in children. Occasionally late cases are met with after the age of sixty. It is a common disease in the great centers of civilization, where the intellectual stresses are most severe, and is comparatively rare among lower races. For instance, it is seldom observed among the native Egyptians or Icelanders. The disease is more frequent among men of ability in professional or business life than among the ignorant and uncultured.

As regards the position of syphilis as an etiological factor, it may be said that a certain history of syphilis is obtainable in at least fifty per cent. of the cases, and it is probable that the true relation is considerably larger. Several years ago, in a study of this subject, I examined the contributions of no fewer than seventy authors to the elucidation of this problem. There was wide divergence in the statistics presented; but from my examination of all these figures, it was assumed that between sixty and seventy per cent. of all cases of general paralysis were syphilitic, if not far more.

By a comparison of statistics of the relation of syphilis to all other forms of insanity, which I have estimated to be from six to ten per cent., we have the further fact that syphilis is seven to ten times as frequent in dementia paralytica as in insanity in general.

The fact is thus established beyond dispute that syphilis is a striking etiological factor in general paresis, and many modern authorities have come to look upon it as a true parasyphilitic disease.

A much more difficult problem is to determine the exact nature of the relationship between syphilis and general paresis. Is it a direct cause, or merely a contributing agent? Is it in syphilitic cases a post-syphilitic affection, or is foregone syphilis merely a predisposing factor? The problem may be examined from several standpoints. In the first place, we have the rather remarkable statistics of Lewin of 20,000 cases of syphilis, one per cent. of which became insane, and in which not a single case of general paresis developed. Then we have the further fact, to which I have already alluded, that among the native Egyptians, where syphilis is one of the most wide-spread of disorders, scarcely a case of general paresis has been reported; and in the asylums at Cairo, which I visited a few years ago, not one such case was to be found. It is significant, by the way, that alcoholism is seldom or never observed among them, the drinking of spirituous liquors being interdicted by the Koran. Such facts as these it is impossible to reconcile with a hypothesis ascribing to syphilis the direct causation of paralytic dementia.

Again, from the pathological standpoint, it is well known that the direct invasion of the brain by syphilis is characterized by changes in the blood-vessels (endarteritis obliterans), by the formation of gummata, or by diffuse meningeal infiltration (specific leptomeningitis or meningo-encephalitis). The first and third of these processes are most frequent in and about the base of the brain. The second is more common in cortical regions. On the other hand, in general paralysis we have a chronic meningitis of the convexity with atrophy of the cortex, and the processes in this disease and in syphilis are quite distinct, although there are cases in which a syphilitic meningo-encephalitis may closely simulate symptomatically dementia paralytica. The pathological processes are different.

The best recent English exposition of the relationship of syphilis to general paresis is by Dr. Frederick Mott, in the "Arch. of Neurology," published by the Pathological Laboratory of the London County Asylums, Vol. I. He does not consider the dictum "no syphilis, no general paralysis," proven, but believes all the evidence is in favor of the strong influence of syphilis in its production, and brings forward this evidence in a convincing way. One of the most cogent reasons is to be found in his study of twenty-two cases of juvenile general paralysis occurring from the age of eight to twenty-three years. Syphilis could not be excluded in any of these, but was positively determined to be present in thirteen cases, and in the parents in four, making a total of eighty per cent. He feels, however, that all were syphilitic. Dr. Mott considers the pathological process in both tabes and general paralysis to be identical, and agrees with Möbius, who calls them metasyphilitic, the same disease affecting different parts of the nervous system. Since my own studies of this subject in 1892, I have devoted more than usual attention to an extremely careful examination of the history and of the bodily conditions in paretics in whom syphilis was denied, and feel sure that the percentage is much higher than the figures given by me at that time. There are still some few cases in which it is not possible to discover, either upon the person or in the history, any evidence of previous syphilis, but we know that patients are often seen in whom the specific infection has been so mild in character that no residua are left upon their persons, and they have no memory of initial lesions, yet the conditions are such that they must have had syphilis. As Mott says, women, for instance, are often infected and have no characteristic lesions. They may be immunized by a spermatically infected fetus, so that they have the disease in so mild a form that there is no noticeable impairment of health.

Alcohol would seem to be a factor in some twenty per cent. of paretics. Other toxic agents (lead, tobacco, rheumatism, etc.) are also believed to take a part at times in the etiology. Trauma has often been mentioned as an occasional cause of paresis, but there is no well-authenticated instance in literature of such etiology, and until better evidence is offered we must doubt the sufficiency of this factor.

In most cases, as already intimated, several of the causes named are associated in the production of the disease.

Symptomatology.—The disease is best studied in its three stages —the prodromal period, the established disorder (which may be exalted, depressed, or hallucinatory), and the terminal period of dementia.

Prodromal Period.—General paresis is one of the most insidious forms of insanity as regards its gradual, almost unnoticeable onset. Very often this early stage presents symptoms which lead to its being mistaken for neurasthenia. Indeed, the earliest symptoms may be neurasthenic in character, or even a combination of hysteria with neurasthenia. Sleeplessness, tremor, irritability of mood, hypochondriacal depression, dull headache, ophthalmic migraine, pains in various parts of the body, general malaise, loss of appetite, and digestive disorders— these are the manifestations which may be readily misinterpreted as purely of functional nature. It is only when other symptoms in addition to these are presented that a suspicion of a more serious malady may be entertained or the diagnosis actually established. These symptoms are, on the mental side: little faults of memory; errors in speech or writing; the misuse of words; the leaving out of letters, syllables, or words, or their reduplication in writing; growing indifference to the higher sentiments; loss of the critical faculty; small lapses in the proprieties, and failure of interest in the more important affairs of life. As these mental features become more and more pronounced, the patient loses and mislays things, makes mistakes in money matters, errs in appointments, confuses persons and objects, forgets his way, becomes easily angered, markedly offends the proprieties, shows extravagance in the use of money, evinces distinct loss of the ethical feelings, exhibits proclivities to sexual and alcoholic excess, and becomes negligent of his dress.

In the earlier period the patient, like any neurasthenic, has a distinct consciousness of his own illness and observes his symptoms. But with the progress of the malady—and it is in this that we find an important contrast to the course of neurasthenia—he loses that sense of being ill, takes no further notice of his own symptoms. On the physical side there are a number of significant marks which are helpful in making an early diagnosis: defective innervation of one side of the face, causing a slight paralysis; transitory ocular palsies, diminished sensibility to pain, Argyll-Robertson pupils; diminished, lost, or exaggerated tendon-reflexes; a dark, pale, greasy complexion; lack of facial expression; jerky tremor of the faciolingual muscles at the beginning of voluntary movement; slight difficulties of articulation; rushings of blood to the head, and attacks of syncope or of mild or severe epileptiform convulsions. A number of other early symptoms have been described by various authors to which some value attaches: loss of memory of localization of tactile sensations (Ziehen); loss of the cremasteric reflex; testicular insensibility; peculiar respiration, with short inspirations, followed from time to time by prolonged sighing expirations (Régis); gastric and vesical crises (Hurd); calcification of the sternum, with incurvation of the xiphoid appendix and consequent interference with thoracic breathing (Régis).

Period of Establishment of the Disease.—When the disorder is fully established after a prodromal period which may range over months or years, it is marked by both physical and mental symptoms which are usually characteristic:

Chief Physical Symptoms.—(1) Peculiar articulation and writing—the "paretic speech" and "paretic writing"; (2) tremor; (3) pupillary disorders; (4) lost or exaggerated tendon-reflexes; (5) muscular weakness; (6) apoplectiform and epileptiform crises; (7) emaciation; (8) trophic disorders.

Mental Symptoms.—(1) Failure of memory for both recent and old events; (2) diminishing number of concrete, abstract, special and general ideas; (3) weakening of judgment; (4) loss of sense of time and place (lack of orientation); (5) delusions (marked by enormous exaggeration, whether exalted or depressed); (6) hallucinations and illusions; (7) emotional irritability; (8) exalted, sometimes depressed, mood; (9) loss of ethical and esthetic feeling.

We will now examine these symptoms somewhat in detail.

The paretic speech is so characteristic that, heard a few times, it is always remembered; yet it is difficult to describe. There are shades of difference in various individuals, so that authors qualify the disorder of speech as drawling, stammering, hesitating, scanning, spasmodic, ataxic, and so on. It has some resemblance to the speech of a drunken man. Doubtless the main seat of the lesion affecting the speech of the paretic is in the cortical motor speech-center, but sometimes the lesion is probably in the bulbar centers connected with the elaboration of the motor impulses requisite to articulation. The jerky tremor or ataxia of the speech-muscles, together with incoördinated impulses from the cortical motor speech-center, is responsible for the peculiarities in speech. Labials and certain consonants are the most difficult for the paretic to enunciate, and the typical speech is shown in the attempt to pronounce such words or phrases as "electricity," "artillery and cavalry brigade," "immovability," etc., in which the consonants may be left out, drawled over, misplaced, or even reduplicated thus: "electericity," "artillililery," "bigrade," "immobilty." As the disease advances, the words are run more and more together, until finally the speech is utterly incomprehensible.

The handwriting of the patient is of equal, and in the earliest stages even of greater, importance. Lapses of words, repetitions of words or even sentences, and especially elisions and reduplications of letters or syllables are extremely significant.

The tremor in paretics affects all parts of the body, but is especially noteworthy in the face and tongue. In the tongue it often takes on a fine, fibrillary character. It is very rare in even pronounced neurasthenic conditions to observe tremor of the facial muscles. Still we do meet with it at times, and the distinction that I would draw between the facial tremor of profound neurasthenia and that of paresis is that in the latter disorder there is a peculiar jerkiness and ataxia in the tremor, especially at the beginning of a voluntary movement. Thus, in asking the paretic to wrinkle his forehead, an ataxic tremor will be set up in the occipitofrontalis. In snarling up the nose, it is observed in the small muscles about the cheek and nose. In showing the teeth, the ataxic tremor becomes marked in the levators of the lip. In protruding the tongue, there is a rapid, jerky tremor at the beginning of the movement.

As regards the pupils, the most important sign is absence of the reflex to light. Next in order comes extreme miosis (pin-hole pupils), and next in importance a variable inequality (one pupil being larger at one time and the other at another time). Irregularity of outline of either or both pupils is significant. Simple inequality of the pupils is less distinctive because met with in other forms of insanity, and occasionally in normal persons. Marked mydriasis is very common in the latest stage of the disease.

In tabic forms of the disorder the knee-jerks are diminished or lost. In all other forms the tendon-reflexes are apt to be enormously exaggerated, so that we get not only extreme knee-jerks, but quadriceps clonus, ankle-clonus, jaw-jerk, jaw-clonus, and extreme wrist- and elbow-jerks. With this spastic condition we observe also considerable rigidity of the muscles, with a tendency in the latest stage to marked contractures. Often in tabic forms, when the knee-jerks are at first lost, they become finally exaggerated. Hence, while the term tabic is often used to describe a form of paresis in which we have lost or diminished knee-jerks, together with Argyll-Robertson pupils, this is simply a descriptive designation, and does not necessarily imply that we have a combination of locomotor ataxia with paresis.

As previously stated, one of the chief symptoms of paralytic dementia is a progressive weakening of the muscles in general of the whole body. It is rather an enfeeblement than a paralysis. It is manifested mainly by localized pareses in various muscles or groups of muscles. These are often noted as early symptoms—for instance, in the eyes and face. In fully one-half of the cases we observe, at one time or another, weakness of some of the ocular muscles, not infrequently giving rise to diplopia or ptosis, rarely nystagmus. A certain amount of ptosis is often seen, and the overaction of the occipitofrontalis in consequence forms a striking picture in many cases. One-sided paresis of the forehead muscle, orbicularis palpebrarum, or lower face is rather common. The muscles about the mouth are particularly often involved, so that marked inequality of the nasolabial fold and of all of the oral movements is encountered. The speech has frequently a nasal tone from one-sided or double palate paralysis. Deviation of the tongue is common. The general strength of the extremities, as measured by dynamometers, is diminished, sometimes on one side more than on the other, presenting the picture of a hemiparesis. The want of equal innervation is sometimes indicated by the attitude of the patient, the inclination of the body to one side or another, backward or forward, sinking of the head on the breast, etc. Weakness in the muscles of deglutition leads to difficulty in swallowing. The peculiarity of most of these paralytic phenomena is, in the first place, their mildness of degree, and, in the second, their frequently transitory character (the weakness may be first on one side of the face, then on the other, now about one eye, now in an extremity, etc.).

Nearly every case of general paresis exhibits, at some time in its course, convulsive or apoplectiform seizures. Usually these critical episodes occur at the height of the disorder or in its final stages, but occasionally they are among the very earliest symptoms. For instance, one case that came under my observation began with a transitory hemiplegia following an apoplectiform attack. Up to the day before this seizure he had performed his difficult duties as an accountant in a large railroad organization to the perfect satisfaction of his superiors, and none of his family had observed any indication of prodromal symptoms. He died as a typical paretic a year later. Another case, much the same in many ways, began with general epileptiform convulsions extending over twenty-four hours. The attacks may appear in the form of syncope, or coma, or aphasia. A peculiarity of all of these crises is their transient character, and as even in cases terminating fatally in such attacks often no lesion has been found, their pathogeny has been ascribed to congestive conditions or to circumscribed edemata in various areas of the brain. As a rule, mental failure becomes more apparent after these crises.

Rapid emaciation is usual after the disorder has actually set in,—that is, at the termination of the prodromal period,—but later on, after the climax has been reached and dementia becomes more apparent, patients often gain largely in flesh.

Among the trophic disorders we note especially bed-sores, which appertain mostly to the terminal condition. In some of the cases a true trophoneurosis is the cause, and in others weakened peripheral circulation and uncleanliness. A striking fragility of the bones is common in general paresis, which accounts for numerous accidents in asylums, such

There are cases which run their course without delusions, the symptoms then being merely the progressive dementia with advancing physical debility. But in a considerable proportion of paretics delusions are manifested, usually of grandiose character, associated with more or less ideomotor excitement (sometimes approaching the maniacal condition), and occasionally of melancholy character. The grandiose ideas of male patients are concerned with wealth, power, glory, size, strength, position, possessions, and of female patients with dress, finery, jewels, and children. At an early period these grandiose ideas are not to be distinguished from the similar fancies of many cases of ordinary acute mania. But when the judgment becomes weakened, as it inevitably does, a peculiarly distinctive character is given to the paretic's delusions. The grandiose delusions take a magnitude, an enormity, a stupendousness not observed in any other form of insanity. Wealth is counted in decillions of worldfuls of gold. The patient is czar, king, president, queen, God, at the same time. His penis is a mile long, his testicles large diamonds. He will bring the Pacific Ocean over the Andes to make the largest waterfall in the world. He will move the asylum buildings on a road of gold to Washington. He has thousands of wives, every one of whom bears two hundred children nightly. He bestows on his physicians and nurses royal orders, dukedoms, writes them checks for enormous sums of money, etc. When the mood of the patient is hypochondriacal or melancholic, the delusions retain the same element of enormity despite their unhappy contents. He states that he is impoverished by having lost billions of dollars; he is committed to prison for thousands of years; he weeps because he can not do his duty to the nations which he governs; there is some horrible condition of his bowels which requires the most awful of operations, etc.

There are some cases of general paresis which exhibit alternating phases of melancholic depression and ambitious exaltation, and these are described as paralytic dementia of circular type.

Hallucinations and illusions are frequently observed in general paresis. They have more or less relation to the condition of exaltation or depression present and to the delusions manifested. Auditory hallucinations are the most common. They are noted even in the early periods of the disease, but are generally a part of the maximum period. They are absent in the final stage.

Emotional irritability and changeability are generally evident. The patient laughs or weeps easily, and is often readily angered.

The excesses, sexual and alcoholic, lapses of propriety, etc., are significant of loss of esthetic and ethical sensibility. He indulges himself freely and without morality (though previously moral), drinks immoderately, steals, and squanders his own and others' property. As his character sinks lower and lower he commits all sorts of shameless offenses against decency.

Before passing on to the final stage, we not infrequently encounter, in the course of the disorder, peculiar interludes of recession of all of the symptoms. These are known as remissions. Remissions last from several weeks to several months, as a rule, occasionally for a year or more. Very striking at times is the remarkable improvement to be observed in a remission. This may attain to a degree making it almost impossible to discover any vestige of deviation from the patient's normal mental health. The extraordinary delusions disappear, the maniacal or melancholic mood vanishes, the symptoms of confusion and forgetfulness pass away, and noteworthy intellectual lacunæ are filled again. The patient may return to his affairs. It is very rarely that marked physical stigmata of the disorder diminish and give place to normal conditions. After a time the old symptoms of the dread malady reassert themselves and its fatal progress is rebegun.

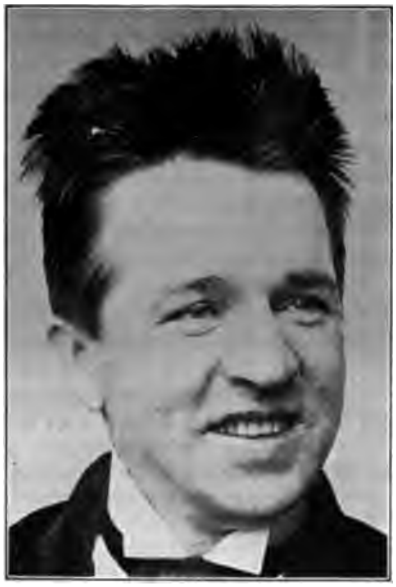

Fig. 312.—A noted actor who recently died of paresis. Taken to show the expression of paralytic dementia in an unusually expressive face (loaned by Dr. Atwood).

Terminal Period.—As already intimated, there are cases in which there is merely a progressive enfeeblement of mind and paresis of body from beginning to end, with none of the excited or depressed conditions, delusions, hallucinations, remissions, etc., just described; cases which pass by gradual stages from the prodromal into the terminal period. In the main, however, we have most of these other manifestations interpolated. The final stage is often ushered in by the convulsive or apoplectiform seizures. This is the stage of more or less complete dementia. We may still note the remains of old grandiose or hypochondriacal delusions in the scarcely comprehensible mumblings of the paretic dement, but usually the mind becomes completely vacuous; the patient speechless, filthy in his habits, bedridden, and more helpless than an infant. He lies in bed, either motionless or restlessly moving his limbs and grinding his teeth. He can scarcely swallow his food, and often requires to be fed to prevent strangling. He wets and soils himself, and bed-sores and contractures develop. Finally, death by inhalation-pneumonia, septicemia (from the bed-sores), cystitis, marasmus, intestinal catarrh, or exhaustion steps in to draw the curtain on the distressing picture. Not a few die at an earlier period in an epileptiform or apoplectiform crisis.

Duration and Prognosis.—Paralytic dementia runs its course in three to five years, on an average. There are more cases which terminate under three years than over five, but cases lasting five years are not infrequent. A duration of ten years is among the greatest rarities.

The prognosis is practically always death within a short term of years. The author has never known personally of a case recovering. In our whole literature there are, according to Ziehen, but a dozen cases of recovery on record. It is probably questionable if even these were genuine cases of paresis, since an error in diagnosis is not at all uncommon.

Diagnosis.—The chief disorders which may be confused with paralytic dementia during the various stages of its evolution are neurasthenia, alcoholism, syphilis of the central nervous system, acute mania, epileptic dementia, paranoia, or secondary paranoia with delusions of grandeur, multiple sclerosis, and mental conditions associated with common organic lesions of the brain (tumor, hemorrhage, embolism, thrombosis). In atypical cases the diagnosis is often difficult and sometimes even impossible.

As regards neurasthenia, it is only in the prodromal period of general paralysis that differentiation may be difficult. I shall attempt to present in brief, tabular form the distinctive diagnostic points of these two conditions:

GENERAL PARESIS (EARLY PERIOD).	NEURASTHENIA.
Sluggish, immobile, irregular, pin-hole, or unequal pupils.	Large and rather active pupils usually.
Diminished, greatly exaggerated, or unequal knee-jerks.	Active and equal tendon-reflexes.
Fibrillary tremor of tongue; jerky, ataxic tremor of fingers, face, tongue, occipitofrontalis.	Tremor fine and rapid of fingers and eyelids, not jerky, very rarely involving face, almost never the tongue and forehead.
Elision or reduplication of letters, syllables, or words in writing.	Nothing abnormal in the writing.
Sometimes noticeable characteristic defects in speech.	No changes in enunciation.
Usually little or no notice taken by the patient of his symptoms.	Patient pays marked attention to his symptoms.
In some cases a feeling of cheerfulness and well-being out of proportion to the actual disorder present.	Patient apprehensive and alarmed at any symptoms present.
In many cases a vague, hypochondriacal depression with tearfulness, not referred to any definite physical cause.	When hypochondriacal, patient's attention fixed on some definite morbid process which he believes to be going on in his system.
Actual evidence generally found of failing memory, defect of intellectual process, weakened judgment, and loss of esthetic and ethical feeling.	No evidence of mental decay or loss of esthetic and ethical feeling.
Occasionally epileptiform or apoplectiform crises.	Nothing of this kind in neurasthenia.
Vertiginous attacks and transitory aphasia of mild degree.	Not present in neurasthenia.

In chronic alcoholism we may have presented to us many symptoms, such as tremor, thick speech, mental changes and defects, epileptiform crises, and, where rudimentary polyneuritis is present, lost knee-jerks, which may simulate the syndrome of paralytic dementia. The resemblance is sometimes remarkably close. The chief differential point is the great improvement and often recovery which take place in alcoholic mental disorder on withdrawal of the alcohol. With abstinence the speech becomes normal, the tremor grows less or disappears, the knee-jerks return, epileptiform attacks cease, defects of memory are no longer perceptible. If hallucinations are present, they are more often visual and zoöscopic in alcoholism, while generally auditory in paresis. The delusions of the chronic alcoholic are, as a rule, suspicious and persecutory. It must be remembered that a typical general paresis may, however, develop on the basis of a chronic alcoholism.

Aside from the comparison of neurasthenia with the prodromal period, probably the mistaking of syphilis of the central nervous system for advanced general paralysis is the most common error in diagnosis. The two disorders have so much in common that their differentiation is often only possible by prolonged observation through the whole course of the disease; and if the paretic dementia should happen to progress as a simple dementia with none of the characteristic episodes, the diagnosis is sometimes quite impossible. The following table will serve to make some of the similar and unlike features of the two maladies apparent :

GENERAL PARALYSIS.	CEREBROSPINAL SYPHILIS.
Paresis of mild degree of cranial nerves at times. Slow in onset and transitory.	Complete paralysis of one or several cranial nerves often. Generally sudden in onset and stable.
Symptoms of a diffuse general lesion.	Symptoms of multiple lesions.
Jerky and ataxic tremor.	No tremor in syphilis.
Loss of iris reflex to light, preservation of movement of iris in accommodation (Argyll-Robertson pupil); extreme miosis.	Iris often immobile both to light and in accommodation; extreme miosis very infrequent.
Characteristic elisions and reduplications of letters, syllables, or words in writing.	If any change in writing at all, due to agraphia or dementia. No resemblance of the changes to those of paresis.
Peculiar disorder of speech. (G. P. speech.)	No speech disorder usually, but, if any, due to organic aphasias of one kind or another. No resemblance to the G. P. speech.
Headaches vague, transitory, and seldom distressing.	Headaches extremely severe, constant, and worse at night.
No material changes in the fundus.	Optic neuritis occasionally.
Progressive advance of the disease to a speedily fatal termination, with a possible remission in some instances for a brief period.	Irregular advance, with many fluctuations in intensity and character of the symptoms, extending over a long period of years, and not necessarily fatal.
Delusions often expansive, sometimes depressed, characterized by enormous exaggeration in either case.	Delusions rarely present.
Affective state often expansive, sometimes depressed.	Affective state usually depressed or apathetic.
Progressive mental enfeeblement.	Incoherence and thought-inhibition.
Epileptiform and apoplectiform crises in nearly every case, and frequently repeated.	Epileptiform and apoplectiform seizures uncommon, but if they do occur, are generally single, isolated attacks.
Antisyphilitic remedies useless.	Antisyphilitic remedies of marked service.

A gummatous meningitis may, however, present a typical general paresis in all its manifestations, and there are cases in which the actual lesions of paresis exist side by side with syphilitic cerebral lesions.

We may have maniacal outbursts in the course of general paresis. Indeed, I have seen paresis begin in a number of instances as an apparent acute mania. During this maniacal state the chief means of differentiation of the two disorders is in the character of the contents of the delusions. Both are exalted and expansive and tend to the same general exaggeration of feelings of power, strength, intellectual and physical abilities, wealth, social station, etc. But the stupendous exaggeration in general paresis is never observed in acute mania. This is a valuable indication. Naturally, if any of the physical signs of paresis are present, the diagnosis is not difficult.

Epileptic dementia, with its slow speech, mental defect, and epileptic seizures, might at times be mistaken for a paralytic dementia, presenting chiefly these symptoms. But the history of long years of epilepsy preceding the psychic degeneration suffices, as a rule, for the diagnosis. It is only when such history is not obtainable that error might arise.

In paranoia itself, and in paranoia secondary to acute mania or melancholia, the expansive or depressed delusions are of a more fixed and much less exaggerated nature. A study of the character of the delusional contents should make differentiation easy.

Multiple sclerosis, with its jerky tremor, exaggerated reflexes, and mental enfeeblement might at times present a syndrome analogous to that of some cases of paralytic dementia. The tremor of multiple sclerosis, however, while also jerky and ataxic, is a marked intention tremor, exhibiting wider and wider excursions the greater the effort to carry on a voluntary movement. The tremor of paresis, on the other hand, shows no such increasing exaggeration on voluntary efforts to use the muscles. In sclerosis, the head is often involved in the tremor; in paresis, never. Nystagmus, so common in sclerosis, is almost never observed in paresis. The dementia of sclerosis, when present, is slight and not especially progressive, and there are no expansive or depressed delusional episodes, such as characterize paralytic dementia.

Focal brain-lesions (tumor, hemorrhage, softening, etc.) with dementia and paralysis may simulate somewhat certain types of general paralysis, but the progressive character of the latter disorder, with its crises and psychic episodes, should serve to give the condition presented definite outline and character.

Pathological Anatomy.—It is usual to describe the pathological condition underlying paralytic dementia in general terms as a diffuse meningo-encephalitis. The gross changes observed at autopsy are as follows:

1. General diminution of weight of the brain.
2. Increased fluid in the subdural space and in the meshes of the arachnoid (external hydrocephalus).
3. Pachymeningitis hæmorrhagica interna, with large, fresh, or old hematomata of the dura mater (in about half of the cases).
4. Chronic leptomeningitis (opacity and thickening, with adhesion of the membranes to the cortex).

5. Narrowing of the cortex, with gaping of the fissures.
6. Distention of the ventricles with serum and granulated and thickened ependyma (chronic internal hydrocephalus).
7. Gray degeneration in the centrum ovale, brain-axis, in various columns of the spinal cord, and in some of the spinal roots and peripheral nerves.

The microscopical findings may be summarized briefly as follows :

1. Changes in the vascular walls, dilatation of the perivascular spaces, wandering white and red blood-corpuscles.
2. Increase in number of the astrocytes.
3. In the ganglion-cells : loss of the nucleus and nucleolus, cloudy swelling, shrinking of the protoplasmic processes.
4. Degeneration and disappearance of the nerve-fibers with myelin-sheaths, in the white matter and in the cortex, and of the tangential fibers.

The whole cortex is more or less affected, but often the changes are more marked in one area than in another. It is usual to find the frontal lobes especially implicated.

Treatment.—In the majority of cases of general paresis commitment to an asylum is necessary, owing to the dangers arising from the patient's excesses. He may squander his property or scandalize his family by his immoral or criminal acts. It is true that cases which present merely the dual symptomatology of increasing physical debility with progressive mental enfeeblement may be, and often are, treated at home. But, on the whole, it is better to act promptly in placing the patient in a place of safety.

The disease being inevitably fatal, there is little to be advised in the way of medication, save symptomatic treatment. It is quite proper, in cases with a history of syphilis, to try energetic antisyphilitic measures—mercurial inunctions and large doses of iodid. If, by any possibility, there has been any confusion of the malady with cerebral syphilis, this will at least serve to remove any doubt. The opium treatment is of value in the periods of depression, and hyoscin, hyoscyamin, or duboisin (gr. $\frac{1}{100}$ to gr. $\frac{1}{60}$), hypodermatically, in the periods of maniacal excitement. Where epileptiform seizures are frequent the bromids are indicated, and in status epilepticus chloral and starch-water per rectum (gr. xv to ℥j of starch water). Chloral combined with morphin is to be recommended in phases marked by hallucinatory excitement.

Little or nothing is to be expected from the many measures advocated by various authors : setons and vesicants to the nape of the neck, painting the neck with iodin, hydrotherapy, physostigmin, ergotin, and trepanation.

Trephining has been resorted to a number of times in the past six or eight years, but seems to have been abandoned as useless. The theory that led to its use was that there might be increased intracranial pressure, but this theory has been discarded for want of evidence.

When dysphagia is present, the patient may require feeding with the tube. In the terminal period of the disorder catheterization and careful efforts at preventing bed-sores are required.

CHAPTER XIV.

PARANOIA.

Synonyms.—Chronic delusional insanity; Progressive systematized insanity; Primare Verrücktheit; old term, "Monomania."

Definition.—Paranoia may be defined as a progressive psychosis founded on a hereditary basis, characterized by an early hypochondriacal stage, followed by a stage of systematization of delusions of persecution which are later transformed into systematized delusions of grandeur. Though hallucinations, especially of hearing, are often present, the cardinal symptom is the elaborate system of fixed delusions.

The hypochondriacal stage is called by Régis "the period of analytic concentration"; the second stage, "the period of delusive explication"; the final stage, "the period of transformation of personality."

Varieties of Paranoia.—There is one typical form of paranoia to which the main portion of this chapter will be devoted, because it is the type which will be most readily recognized by the student and general practitioner. But there are incomplete or immature forms and atypical variations, which the special student of morbid psychology learns in the course of time to distinguish. Thus, many of those eccentric or queer individuals whom we call "cranks" are rudimentary or undeveloped cases of paranoia. Some idea of the varieties of paranoia noted by authorities may be gathered from the attempts at classification by different writers. For instance, French and Italian authors are inclined to divide paranoia into two great groups—viz., (1) degenerative, with original and late subvarieties, according to the period of life at which the insanity develops; (2) psychoneurotic, with primary and secondary subvarieties, according to whether it develops primarily or secondarily to another insanity.

Ziehen classifies paranoia into two great groups, according to the predominance of either delusions or hallucinations—where hallucinations are the most prominent symptom, he terms the psychosis paranoia hallucinatoria; where delusions are preëminent, he denominates it paranoia simplex. Either form may be acute or chronic. Hence he makes four chief types: (1) Paranoia hallucinatoria acuta; (2) Paranoia hallucinatoria chronica; (3) Paranoia simplex acuta; (4) Paranoia simplex chronica.

This last form is the name given by Ziehen to the complete typical form of paranoia which is described in this chapter, and which he describes as having four stages (prodromal, persecutory, expansive, and pseudodemented). Ziehen also specifies several varieties of acute hallucinatory paranoia—viz., the fleeting-idea form, the stuporous, the incoherent, the exalted, and the depressive forms.

Krafft-Ebing makes two great divisions—original paranoia, appearing in early childhood or before puberty, and acquired (*tardive*) paranoia, appearing between the ages of puberty and sixty years. The latter class he subdivides as follows:

(A) Paranoia persecutoria: (1) the typical form; (2) subtype (paranoia sexualis); (3) paranoia querulans.

(B) Paranoia expansiva: (1) paranoia inventoria and reformatoria; (2) paranoia religiosa; (3) paranoia erotica.

Etiology.—Heredity is a more important etiological factor in paranoia than in any other form of insanity. Krafft-Ebing states that he has never seen a case without hereditary taint. Tanzi and Riva found in their cases of paranoia 77 per cent. of heredity and 9.5 per cent. of infantile cerebral disorders, while in the remaining 14 per cent. hereditary factors could not be ascertained, but were not, of course, excluded. It is more common in females than in males. It affects by preference individuals who are even from childhood peculiar, morbid, shy, irritable, mistrustful, and misanthropic. It is very common to find, in cases of paranoia, some of the various stigmata hereditatis described in the chapter on Etiology, such as cranial or facial asymmetry, malformations of the ear or palate, etc.

Symptomatology.—We will examine the symptoms of the different stages in the order of their development. In the *prodromal period*, the hypochondriacal stage or period of subjective analysis, as it has been variously termed, which may have its conception in early childhood, the patient is morbidly shy, peculiar, eccentric, avoids the companionship of others, and is prone to withdraw himself into the solitude of his own thoughts. The physiological commotion of puberty and adolescence, with its inflow into consciousness of innumerable new sensations, its flood of new instincts, powers, ambitions, and ideas, tends to intensify the morbid proclivities already evident. The patient notes his own peculiarities of conduct, and begins to recognize the singularity of many of the somesthetic sensations which come to him—sensations which at this time might well be considered more or less neurasthenic in character: paresthesias of the head, trunk, viscera, and limbs; pains in various parts of the body, tinnitus aurium, sparks and dots before the eyes, and the like. The unnaturalness of these sensations leads to his spending much time in contemplation of them, so that a hypochondriacal complexion is given to his thoughts. To these physical sensibilities are now added a consciousness of difficulty in the concentration of his thoughts; a difficulty in the proper control of the direction and subject matter of his thoughts. He becomes extremely introspective, and, the more he studies the somesthetic sensations brought to his attention, the more he contemplates the phenomena of the uncontrollability of his thoughts, of their rising unbidden from his subliminal consciousness, of the unrestrained constellation of his presentations, the more is he inclined to search for some cause of his morbid condition. At first, like an ordinary hypochondriac, he investigates himself to find a solution of the problem, and, failing in that, he extends the region of his observation to his environment, seeking there the reason of his strange feelings, general disquietude, and morbid stream of thought. He becomes wholly preoccupied with himself. He can not employ himself, either physically or mentally, as he should. He fails in his duties—in everything he undertakes. People seem strange to him in their conduct and in what they say. He grows suspicious and distrustful of everything and everybody. What is done and said by others appears to have some significant relation to himself. People alter in their conduct toward him, look at him curiously, smile sarcastically when he passes, wink at or make signs to one another when he is near; make observations among themselves which, overheard by him, are construed as having a double meaning, as being derogatory to him, reflecting on his character. The more he studies the extraordinary condition of affairs, the more gloomy, solitary, and self-absorbed he becomes. Naturally, the growing alteration in himself really does provoke the notice of others—a fact which tends to intensify his ever-increasing suspiciousness of concealed animosity among those with whom he comes in contact. Many things in his past life rise up in his memory to find a new interpretation in the light of his present general distrust. His physical sensations have become more marked, have taken on a new character, have altered from paresthesias to illusions, and even hallucinations, of general or special sensibility. He feels peculiar general sensations, shooting pains, sudden prickings in his skin. Unusual and unpleasant odors or tastes harass him. Extraordinary sensations flow into consciousness from his genital organs. Much more serious and remarkable, however, are the peculiar changes in his auditory perceptions. At first these are usually confused noises, or roaring and tinkling sounds, with the gradual perversion of sounds and words heard into illusions colored by the suspicious contents of the patient's consciousness; later, actual hallucinations of hearing, which become a fixed and permanent feature of his malady.

The patient now enters into the second or *persecutory period* of paranoia, the period of delusional explication of his troubles. He has arrived at what he conceives to be a logical result of his reasonings, a rational explanation of the distress and affliction he has undergone. Everything he has suffered has been due to the machinations of unknown enemies. The delusions of persecution are at first somewhat confused in character. No particular individual or group of individuals is thus far responsible for the inflictions. It is simply some unknown persons who take pains to manifest ill-will or malevolence toward him. "They" talk against him, call him names, attempt to poison him with gases or by tampering with his food, and try to injure him with electric shocks or by throwing corrosive substances at him. Since wherever the patient may be, wherever he may go, the voices, shocks, poisons, etc., seem to pursue him, he comes to think that no single person could manage so vast a conspiracy. It must be some large aggregation of persons who are concerned in the effort to humiliate, cripple, or destroy him; an aggregation bound together by ties of secrecy, and able to permeate all classes of society. What could such body be but a secret society, an order of Masons or Odd Fellows; some religious or political brotherhood—the Jesuits, Catholics, Protestants, anarchists, or police.

Perhaps some one individual is at the head of the band of plotters, some arch-conspirator, but the work is done by innumerable *aides*, who employ all manner of means and apparatus to accomplish his ruin. This system of persecutory ideas is built up in the most elaborate way, and the more educated the individual suffering from paranoia, the more wonderful the organization and adjustment of the various parts of the delusional system. The persecutory delusions of other forms of psychoses, such as toxic insanity, senile dementia, and melancholia, may have a certain interest and fixity, but those of the paranoiac are woven together like a romance. The relation of the former to the latter is that of the brief sketch to the serial novel. The telephone, the phonograph, telepathy, hypnotism, and other and more mysterious apparatus and phenomena are brought into service by the relentless league. I do not know the origin of Du Maurier's conception of his novel, "Peter Ibbetsen," but I suspect that many of its unique features, especially that of "dreaming true," were suggested by conversations with some well-educated paranoiac in a lunatic asylum.

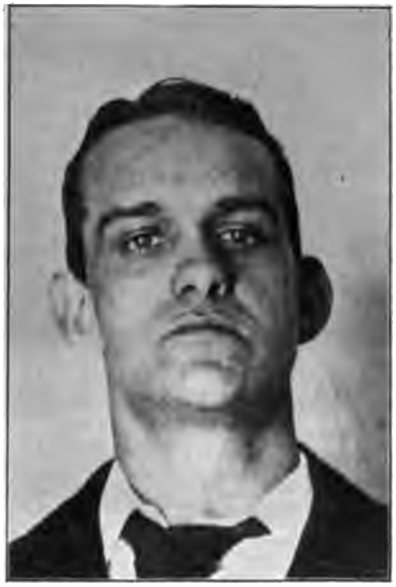

Fig. 313.—Young paranoiac with homicidal tendencies at period of passing from persecutory into grandiose stage (Dr. Atwood).

Many patients seek in a most elaborate way to explain one peculiarity of their auditory hallucinations—viz., the fact that their thoughts are read off by the voice or voices simultaneously with the appearance of the thoughts in consciousness. This adds naturally a new terror to the persecution, for the ability of the conspirators to read off and taunt the patient with his own most secret thoughts is a particularly refined species of deviltry, as well as evidence of the extraordinary psychological power of his tormentors. The voice which speaks his thoughts, or answers his thoughts before he can himself utter them, may be referred to the external world or to some part of his own body. This phenomenon has been variously termed echoing of the thoughts, motor representation of articulation, and verbal psychomotor hallucination. It depends upon the close relation existing from earliest infancy between the auditory word-center and the motor speech-center. Any irritation of this auditory area is immediately, synchronously, irradiated to the motor speech-center. However slight this stimulation of the speech-muscles, recurrent sensations of movement in them are carried back to the brain, giving rise to the hallucinations of internal hearing.

The patient is driven by his delusions to make complaints to the police, to judges, or to the governor of the State, the President, or other government or judicial authorities. Not infrequently he attempts, himself, to wreak vengeance upon one or more of his imaginary enemies. Attempts at homicide are, therefore, common in these cases. The writer had in his charge at the Poughkeepsie Asylum, for some years, Ernest Duborgue, a persecutory paranoiac, who, many years ago, ran through Fourteenth Street, New York, stabbing women right and left with a pair of compasses. More often they seek to escape from their enemies by constant change of residence.

The third stage, the *expansive period,* or the period of transformation of personality, is often induced by the patient's attempt at a logical explanation of the cause of the persecution. Since he has so many enemies, and every man's hand is against him, it must be due to his importance. He either resembles some distinguished personage or he is of royal or god-like descent. The transformation may be suddenly induced by a hallucination revealing to him his high estate. The contents of these delusions of grandeur may be religious, political, erotic, jealous, and so on. For instance, the delusion of being a prophet or a second Messiah is very common (paranoia religiosa). The delusion of being a great discoverer or inventor is frequently met with (paranoia inventoria). Another common delusion is that of being a great social reformer (paranoia reformatoria). A peculiar form is paranoia erotica, in which a person imagines him- or herself to be beloved by some one of superior station. It is a romantic, platonic love in which the patient indulges. He has communications with the object of his delusions, imaginary conversations, through the medium of hallucinations. A good example of this form was that of Dougherty, who followed Mary Anderson all over the country, and was finally sent to an asylum because of his threats to kill any one who interfered with his attempts to gain a personal interview with the famous actress. Measurements which I made of his head showed a pathological excess in the height of the skull.[1] After his commitment to an asylum he shot one of the physicians who had him in charge. Another interesting variety of paranoia is that observed in the litigationists (paranoia querulans), who occasionally distinguish themselves by their lifelong involvement in legal processes (due to an overwhelming egotism, which leads to a continual zealous effort to set themselves right, despite the advice of friends, and the wasting of their property, after the

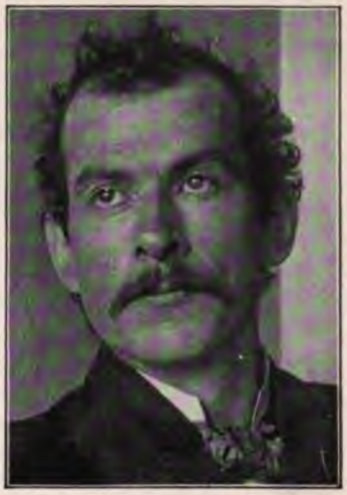

Fig. 314.—Erotic paranoia. "Mary Anderson's lover" (see text).

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[1] "Familiar Forms of Nervous Disease," by M. Allen Starr, New York, 1890. Article on "Paranoia," by F. Peterson, page 299.

ibly trivial lawsuit). Pretenders to thrones, self-styled
, presidents, princes, and so on, are often noted among paranoiacs
ave reached this third stage of evolution. Quite commonly per-
ry ideas still remain in the minds of these patients in association
delusions of grandeur.

n of these periods of development may last for several years,
order may undergo arrest at any period, and there may be varia-
in the degree of development of any stage; so that we constantly
with atypical forms of paranoia. An excellent condition of
nory, judgment, and intellect in all other directions save in those
ted to the single cluster of delusions may coexist. Years ago these
ses were designated as monomania, because of the apparent lucidity
f the patient outside of the limited number of fixed ideas. Many
aranoiacs have distinguished themselves in sacred and profane history,
d even in literature. There have been many of these false prophets
have come to herald a new religion—Mahomet, Swedenborg,
anna Southcott, John of Leyden, John Thom of Canterbury, and
nne d'Arc. We have had them even in the United States within
ew years—the healers exploited by the press. Among political re-
mers we had John Brown and Guiteau. A famous paranoiac immor-
ized himself in his autobiography—Benvenuto Cellini.

I have in my possession a beautifully written manuscript—the
obiography of a paranoiac. He was so dangerously insane that he
t much of his life in the asylum in which he wrote this valua-
work.[1] The volume, bound by himself, is entitled "The Piling
rophet," which is significant of the sufferings he had undergone in
his unhappy life. I believe no better idea of the typical form of paranoia can be obtained than by a careful reading of the history of this case as given by the person himself. It is a graphic picture of the steady evolution of the malady—a remarkable self-dissection of the soul's anatomy. Before presenting the extracts from his autobiography, I shall make a few transcripts from his asylum history.

He was thirty years of age at the time of admission; single; a farm-laborer by occupation. He was not a church-member, had a common-school education, and was a native of the United States. Hereditary predisposition was not acknowledged. His mother, who accompanied him to the hospital, stated that he had always been delicate in his physical constitution, and given to despondency. Since the age of twenty he had done little or nothing, because of ill health. A year previous to his commitment to the hospital as a lunatic he shot himself in the forehead in an ineffectual attempt at suicide. Later, he developed delusions that the people of the village were acting upon him by magnetism, spoke disparagingly of him, and were conspirators against his peace. During the whole of his sojourn in the hospital he had hallucinations of hearing, and in the earlier period of his stay had delusions of persecution. Toward the end of his seven years of hospital life he gradually developed, in addition, delusions of grandeur. Although he had occasional lapses of self-control, manifested by the breaking of window-glass or the tearing of clothing, he was for the greater portion of the time sufficiently self-possessed to restrain whatever violent or destructive inclinations he may have had, and was permitted to go out alone upon the large grounds of the asylum whenever he wished, and to wander about the woods at will.

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[1] "Extracts from the Autobiography of a Paranoiac," edited by Frederick Peterson, "Amer. Jour. of Psychology," January, 1889.

It was during the last two years of his stay at the asylum, while still the victim of constant auditory hallucinations, and of mingled delusions of persecution, unseen agency, and grandeur, that he wrote the volume of four hundred manuscript pages with the extraordinary title of "The Piling of Tophet," this title being founded upon Isaiah xxx, 33. The book itself is a deeper history of his life and mental evolution than any but himself could furnish. It is remarkable for its excellent literary style and for its keen reasoning and psychological analysis of his own disordered mind. In it he dissects his hallucinations and delusions like a skilled anatomist. It is as fascinating as a novel. Every page has its value as an index of the condition of his mind from childhood to the last years of his confinement in the asylum; and the story is told with a directness and simplicity that marks truth upon every statement and lends it such charm as pertains to all works which portray life with the utmost fidelity. In his preface and introduction he makes a diagnosis of his own disease.

Our author, as has already been stated, was not a church-member, and in his book he describes his early religious life and his subsequent beliefs as they developed. His father was a Universalist and his mother a non-professor of religion, although she did attend the Methodist church. During his boyhood he attended the Sunday-school regularly, and at one time the Episcopal church; but his attendance upon divine service ceased in early youth. Both parents were honest, conscientious, and highly respected in the community. They were first cousins. The mother was healthy in mind and body, but the father is reported to have been exceedingly eccentric, possibly insane. From what I subsequently learned regarding him, he also was something of a paranoiac. They strove to bring up their children carefully and to educate them as well as possible.

His father died when the patient was twelve years of age. Up to the age of thirteen he attended a country school both winter and summer, but after that his farm-work permitted him only winter schooling. Still, he evidently had unusual talents and aptitudes, and we find him later studying by himself, in the original, many of the classic Latin authors; and among his favorite companions were the works of Boethius, Lucretius, Josephus, and the Bible. His literary style and modes of thought are in themselves an evidence of more than ordinary attainments in rhetoric, philosophy, and logic.

The matter of heredity in his case was not sifted thoroughly upon his admission to the asylum, nor have I since been able to gather much material relative to this factor in his evolution. But one important element of this nature is described in his book—an element not only hereditary in its character, but for a long time part of his environment, ndoubtedly an influence modifying his mental condition both and after his birth. I allude to a great-uncle, a brother of his nother on his mother's side, who was himself a paranoiac, and who upon the farm in intimate companionship with our patient until tter was twenty-three years old.

we read on we see, from the author's account of himself, how y and environment gradually molded his physical and mental cters. A shy, timid, delicate child; clever intellectually; given to ies of speech and conduct; inclined to solitary musing, rarely shar- the sports or games of other boys—in him were slowly evolved ed eccentricity of demeanor, a disposition to shun his fellows, a nterpretation of their looks and actions as regarded himself, a mor- egotism, a consciousness of a gulf between himself and ordinary n, with deep depression, outbursts of passion, an inclination to icide restrained but feebly by his weakened will, and delusions of ecution. No doubt the derogatory remarks he fancied expressed ut him in the stores were the first harbingers of auditory hallucina- s. Later, he had murder in his thoughts, through the morbid hu- tion he felt at the imaginary insults from others. No doubt, as conduct grew more and more strange, he did attract attention among fellow-men, and this, unfortunately, would but feed the flame of his ological self-consciousness.

We follow his history from infancy through childhood and youth to hood, and observe how, slowly but surely, the hereditary seed sown degenerative soil took root and flourished. His peculiar auditory acuteness, with his morbid shyness, soon gave rise to illusions of hearing, and these again were transformed into hallucinations, as is evident if the thread of the narrative is carefully followed. The curious foundation of his hallucinations he well illustrates and understands. An idea arises in his own mind of what people would say in discussing him, and immediately consciousness in the auditory area projects the idea in spoken words into the environment. He noted this peculiarity of his own thoughts being repeated to him by the voices about him, yet he could not correct the delusions to which they gave origin, but interpreted the matter with the reason and judgment of an insane mind. He naturally had the delusion, founded upon his hallucinations, that people were persecuting him, but upon this now grew another delusion. He began to believe that they could read and repeat his thoughts; that there was some magnetic means by which his tormentors could draw off his thoughts; that other wills could act upon his body, dominating his own will and causing him to do things he had no desire or intention of doing.

It was about this time that he was removed to the asylum. Several chapters of his book are devoted to a description of his life there, his religious beliefs, illusions, and hallucinations. A short time previous to his departure for the asylum he began to read much in the Bible, and, as he says, noted passages which seemed to have a special bearing as regarded himself. There were several coincidences of this kind, and he looked upon them at first as merely coincidences, but in time the resemblance became so strongly marked, to his disordered intelligence, that he came to look upon whole chapters of the Bible as referring to himself. From this the step was not a great one to the delusion of being a prophet. In reading we find that our author had several incentives for writing this book. It contains the autobiography of a new prophet, as well as the revelation of a new religion. From his standpoint, as a man in whose destiny are wrapped up the destinies of the world, he tells posterity of the tortures and trials he has passed through as an atonement for the sins of the earth; how he was mocked and scoffed at, his brain acted upon by magnetic agency, and himself imprisoned in a lunatic asylum for years. Hence the title of his book, "The Piling of Tophet." But behind this insane egotism there shines at times some faint glimmer of the truth, so that he frequently speaks of himself in the terms used by his fellows, as insane, a lunatic, a monomaniac, as having hallucinations; and he thinks the opinions of his friends, relatives, and physicians of sufficient worth to merit considerable argument in his book. He knows what insanity is; he recognizes it in his asylum associates. He could at times "see the man he ought to have become rising up like a shadowy phantom in judgment on the wreck he really was." But this occasional consciousness of their disordered mental condition is by no means infrequent in the insane.

Shortly after writing his autobiography he was removed to a county asylum, where he remained, without change in his mental condition, for several years, when his friends took him out to live with them. He died a religious paranoiac in 1886. He did not become completely imbecile, as such cases often do; nor did he write any further articles, so far as I am aware. Doubtless the indifference with which the world received the propagandism of the new prophet caused his philosophical withdrawal from active warfare in the fields of reform and theology.

In the preface he defines the scope of the book as follows:

> "This work is given to the public as a lunatic's defense of his position. Every effort I have made hitherto to come to an understanding with my fellow-men, on things which I see to proceed from them, and which give my life its whole shape, has drawn out nothing more than blank denials of all knowledge of the things I spoke of. Now, it is impossible for me to reduce my thoughts to the bounds which others have been willing to concede. The object of this little autobiography is to show the form and consistency of the thought that is in my mind.
>
> "I present my evidence to the tribunals of last resort, the public and the press, and ask them to try the case and render their verdict. Have I a right to my thought, or have I not? If not, where am I deceived? If I have, why is not mine the true thought for all men?"

A paragraph from the introduction further reveals the object of his confessions:

> "A person is supposed to have a reason for what he does, and I might consider it incumbent upon me to tell the motives which actuate me in thus entering upon the work of the scribe under circumstances so peculiar. Is there anything I have to tell that might not as well and

tirely be left untold? It is a question which I do not have to ler and decide to-day, for I have been long inspired with the con- ie consciousness, that I have something to tell that it would be world's while to hear."

another introductory paragraph he makes an excellent diagnosis. mental infirmity. Addressing his reader, he says:

"I did not tell you that I am a patient in an asylum. I am to take or granted at the outset that my prospective reader knows nothing of character, condition, or circumstances beyond what I tell him. I here as an insane patient. I have been here over five years. . . . ng an insane man, it will be nothing unexpected that I should, in ing these reports of my fortunes, narrate incidents and particulars taking more or less of the marvelous or preternatural. I am not ly a lunatic, but one of the class of lunatics having a controversy with ie world in general; in other words, possessed with a monomania, or azy one-sidedly or on a single subject."

In the hospital record presented above, nothing is adduced as to redity in this case, and but little stated concerning his mental condi- on in early youth. These deficiencies are, to a great extent, supplied the autobiography. I shall permit our author first to describe his earance in this world, in a cyanotic condition, and the characteristics his childhood and early youth, and subsequently the hereditary uence in his destiny:

"It is said that I was entirely black when I was ushered into the world, and that for I forget how long a period of time I did nothing but give vent to heart-saddening wails. Was I lamenting the gift of light, on this morning of what was to become a woe-burdened existence?

"I was a weakly infant. I came near dying of the whooping-cough, and it was always asserted, by those who knew, that I owed my life to the untiring exertions of a poor woman who lived a neighbor, who busied herself all night with me, dipping me at intervals into a tub of warm water. My half-sister had it at the same time and died.

"It will be of use to give an idea of my nature and disposition in my tender years. I was always a shy, retiring child; not disposed to make free with strangers; not much given to prattle—in fact, one of the sad and silent sort from the first. I can remember some peculiar sensations which used to weigh on my mind, which go to show that the foundation of my mind-life was but imperfect from the first. I used to be troubled with very strange feelings when I was waking out of sleep, especially if I had been taking a nap in the day-time. It used to seem to me that I was floating in the air, and I often thought to myself: 'Why, how queer I have been feeling!' It was as if I filled the whole room, way up to the ceiling. I was told by others that I sometimes raised myself up in bed after getting to sleep and made an outcry, 'Oh, don't! Oh, don't!' seeming to be in great distress; but the strange part of it is that I could remember nothing about it. I do not think that I ever remembered even their waking me, or finding them at my bedside. I only had their word for it next day.

"As far as I can go back, I remember having at times, but not frequently, impressions which must be identical with what I have lately heard others speak of as 'double memory.' The feeling would all at once creep over me that the very thing I was present with, my ideas and perceptions at that time, had happened to me once before in just the same sequence and arrangement. I have heard this explained as due to a lack of simultaneity in the action of the two lobes of the brain, the tardy one remembering what had already passed through the other. My own theory was different, leaving the organ acting out of consideration. I only went so far as to look at it as a mistaken quality in the perception—an erroneous attaching of the nature of the act of remembering to what was really the act of thinking in the present.

"I was very early in life an observer of my own mental peculiarities, to a degree which I think must be a very rare exception. I often used to be sensible of an unsatisfactoriness in my consciousness of what surrounded me. I used to ask myself, 'Why is it that while I see and hear and feel everything perfectly, it nevertheless does not seem real to me? It is as if I were in danger of forgetting myself and the place where I am!' I often wondered even how I kept the run of things as well as I did. I always found myself holding on to the orderly and proper connection of my acts, and yet from my feelings I could not have answered for my doing so. I can remember sitting at my desk in school, when a small boy, and dwelling with melancholy on this dimness in my perception of existence, and wondering how it was with others in this respect. I wondered to myself if life, as ordinarily bestowed, included this deficiency.

"I showed in my tastes and behavior a harmony with the internal composition of my mind. I was never given to the active sports which the common run of boys take so much delight in.

"The simple fact is that I had a languid nervous development, and from the necessity of my organization could not have much capacity or relish for sports of agility.

"If I could compound a boy of my own I should try to improve on the model I remember to have exhibited in myself.

"It is not true that I was regarded or treated as strange or deficient in my wits. Such an idea would look misplaced to those who knew me and consorted with me in those days. These differences are perhaps more evident to myself than they ever were to the greater part of my acquaintances. I brooded on this side of my character at a later period, and I no doubt remain liable to give greater prominence to disparaging traits than some impartial observers would justify me in doing.

"As a general rule, my harmless and peaceable disposition kept me out of squabbles with my schoolmates. If I was approached in an aggressive way, I met it with absolute non-resistance, which in my case had the disarming effect which is attributed to it by pious moralists.

"If we change the scene from the playground to the schoolroom, we shall find that I attained a distinction of my own, apart from the average, and more to my advantage there. I was always a favorite with my teachers. I never gave them any trouble, and took to my studies with a willing relish that could not but be pleasing to them. I learned to read before I went to school; in fact, like an old asylum acquaintance, Mr. M., inventor and infidel monomaniac, I can almost say that I can't remember when I could not read.

"I was frequently singled out for complimentary remarks on my studies. I gave evidence of some talents of a higher —could draw, for instance, better than any boy in the school.

"One of the most marked weaknesses of my character, as a child, my susceptibility to being teased.

"After having pondered some on the traits of the human animal in particular, I have come to the conclusion that there is no further lanation needed than that the impression made on the teaser by the able is such as to naturally prompt the acts constituting the teasing, the sense of burning makes us shrink, and an aroma suggestive of a e flavor tempts us to bite. I feel convinced that the liability to be sed rests on a principle that has a mighty influence in the motions of soul of humanity.

"My misdeeds, as a child, were rarely prompted by a love of mischief or the result of headlong thoughtlessness.

"I had a well-defined idea of the nature of sin, and I used frequently at night to recall the events of the day, and reflect on instances in which I had transgressed and given way to ill-humor, and form resolutions to try and do better. From some of the most flagrant of the sins and improprieties to which small and larger boys are prone I was entirely free.

"My early training can not be said to have been a predominantly religious one. My mind was neither imbued with ineradicable prejudices nor prepared for reaction to the other extreme by excessively rigid sectarian drilling and formalism.

"I worked steadily upon the farm, though with moderation, at such kinds of work as I seemed to be equal to. The heavier kinds of work, such as plowing and wagoning, as also the marketing of the produce, were attended to by my great-uncle.

"It is a somewhat delicate subject to manage to my satisfaction this that I am about to enter upon, but it demands candid and impartial treatment, because the events that followed in later years can not be rightly understood without it. It is impossible for me to give a veracious sketch of my soul-life during this period without dwelling quite minutely on the characteristics of my great-uncle. He was a man who had roughed it a good deal in the world, had been at one time in his life a live-oaker in Florida. How his temper and disposition may have been at an earlier period I can not say—I only remember him as a man possessed of the belief that a certain young man living on an adjoining farm had the power to torture him at his pleasure, both by bothering his brains and inflicting physical pain; which power he made use of to such good effect that the poor victim was almost constantly kept busy holding him at bay by means of cursings of the most fierce and vigorous description. While at work with the horses in the fields, and when driving, he would intermix his commands to the animals with savage execrations of the troubler of his peace. The unfortunate man was troubled, at certain seasons of the year especially, with sore feet, and at such times his imprecations against the offender would fairly rise to yells, and were almost blood-curdling in their intense ferocity. Thus it went on day and night. He slept in a small room in one of the outbuildings, and often he could be heard at a great distance off shouting out threats, sometimes throwing boots or boot-jacks against the boarded side of the building where he lodged to put in the interjection points.

"It may be imagined that a boy of a reserved and sensitive disposition, as I was, could not assimilate very well with such a character as this. I was always distant in my intercourse with him, and a feeling of aversion for his habits of savagery led me to avoid coming in contact with him more than was rendered necessary by our joint labors on the farm.

"As the years passed on and I continued to live in the presence of my uncle's fierce demonstrations of hostility against the invisible destroyer of his comfort, my tolerance for his conduct insensibly gave way. I had now reached the age of eighteen or nineteen; was a tall, slender youth, not strong either in nerve or muscle.

"The exhibition of his ruling passion called up more and more determined feelings of antagonism in my breast.

"Before I knew it I had gone a criminal length in my resentful feeling. I came at last to feel that a person of such a thoroughly savage character did not deserve more indulgence than a mad dog. My position from that time was one of contingent murder. Alas! that I should have been content to let such a state of things last a single day. The frightful danger of my situation ought to have been sufficient to spur me to sacrifice everything to escape from it. But I was in chains, the chains of apathy, impotence, and incapacity, and I could only stay where I was and fume against the object of my detestation.

"I must always regard it as one of the most unfortunate things in my unfortunate career that I should have been placed in contact with this much to be commiserated sufferer at such a time of life. It was not the man himself that I hated. When my judgment could act without impediment, I saw that his unpleasant behavior was entirely the phenomena presented by his never-ending war against what was, in his eyes, the most wicked and cruel of persecutions. I could then pity him and dismiss all rancorous thoughts."

This antipathy led to a change in the residence of our author. He felt that he must be separated from his uncle, and, accordingly, he removed to a town at some distance from the farm. It is curious that he never speaks of his uncle as insane, and it is probable that both his mother and himself and other relatives regarded his persecutory delusions as merely evidence of eccentricity. Soon after removing to town he had some pulmonary difficulty, and he speaks at some length of this as follows:

"In the depressed state of my nerves I imagined myself much worse than I really was, and, like many others in the same condition, I felt as if I was liable to sink away and die at any time. My disease was accompanied with periodical accesses of fever, and in the fictitious strength of excitement given by this my mind seemed to gain an abnormal activity. It was at this time that I first received a revelation on the mysteries of the human soul that had an all-dominant effect on my destinies and the turn of my thoughts ever after. . . . I now learned what had always been to me a hidden mystery—what was the meaning of strength of will and strength of intellect. Before, I had ever lived enshrouded in mists and clouds. In that transitory strength given by the fever coursing through my veins, I now saw the man I ought to have become rising up like a shadowy phantom in judgment on the wreck which I really was . . . My agitation was so great that my mother and the neighbors seemed to fear that I was going crazy. *I* felt that I *had* been crazy for a long while and had just recovered reason. It was a fact. But I was constrained to lock up my remorseful agony in my own breast."

We have seen that our patient was throughout his early youth morbidly subjective, and his hypochondriasis increased with years. He had now attained the age of twenty-three; we shall let him describe his mental condition and habits of life at this time. In this description we shall see the gradual growth of persecutory ideas upon a favorable soil:

"My strength and endurance were not sufficient for manual labor, and I did not feel confidence enough in the clearness and energy of my mind to justify me in making application for any post where head-work would have been demanded, or for which ready presence of mind or a good address would have been required. But it was the unpleasantness felt on contact with my fellow-men that operated more strongly than anything else in binding me down to the course of life to which I devoted myself. I felt my deficiencies most keenly every time I met a human being face to face. . . . . I could not do otherwise than shun what was so galling to my sensibility, while appearing to conduce to no desirable end. . . . . But I am going to show that I still remained exposed to very great dangers, and it is as true as it was before that I shunned the only means of averting the calamities threatening me, no doubt of necessity at this stage, and in obedience to the eternal decree that every tree shall spread out and develop in accordance with the qualities given to it 'before it was in the ground.' I did not like the constraint imposed upon me by the presence of man. I did like the freedom of solitude. I strongly disliked many things I noticed in the manner and words of some I met, and there was nothing to prevent this dislike from occasionally being absorbed into my solitary musings, to find its final resolution in the passion of indignation in its various degrees of intensity as the case might be. I have spoken before of my defective means of defense against 'teasing' or mocking for the purpose of troubling. I was always terribly alert and sensitive to all kinds of 'snubs' and sneers, and oblique remarks in general, on their proficiency in which some people pride themselves so much. . . . . I was also disagreeably impressed by the ways of some who showed a disposition to turn their attention to myself, instead of confining themselves to the subject I was presenting to them.

"I was being carried into a state of secret enmity to mankind in general by the prevailing tenor of my brooding meditations, and there was no corrective present.

"But all received a hue from a yearning for what was worthy in life, paired with a mournful sense of its hopeless absence. Whatever wrong turns I may in my weakness have been betrayed into, it is impossible that I should look upon my then existing frame of mind as a whole with repentant feelings. As well condemn righteousness and holiness itself!

"When I admit that I occasionally was overcome with an irruption of hard feelings toward wrong-doing man, it will, of course, not be understood that I was habitually morose and spiteful in temper.

Nothing could be further from the truth. What commotion there was was mostly internal, rarely reaching the surface in visible ebullitions. . . . . I occupied myself with the trifling labors of my garden, dwelling with interest and pleasure on the progress of my crops and flowers, and every now and then took a ramble over to the woods lying to the south, which were a favorite place of resort to me all the while I lived there. There I botanized and moralized, explored the recesses of the woods, enjoyed the calm quiet of nature, and groaned over my hapless condition, wondering what it was to come to.

"There were some little things that happened to me the first year after I left the farm which became, as it were, a kind of sample of what I must continue to expect, and the memory of which had more influence over my action in after time than I was aware of myself, no doubt. . . . . When I was around the city, thinking I might get employment I called on one of my old acquaintances, who was then in a store. I talked with him a few minutes at that time. I called again a short time after, when I was told by the proprietor that the gentleman I had called to see was not in. There were a number of men present in the store,—salesmen,—and it became apparent to me that they were trying to exhibit an offensive demeanor toward me, or perhaps it would be as true to say that they were moved to make a derisive demonstration against me. At all events, all, with perhaps the exception of the proprietor, stood with contortions of countenance, which was perhaps laughter, until I retired. . . . . I found it hard to consign this to forgetfulness. At first it lay dormant, but it would come up, and I must confess I had hard feelings, even revengeful feelings, toward the actors. Another thing happened the same fall. I went to a store, and, standing at the counter, was noticed by one of the clerks,—an Irishman,—who came to me and said, 'I always wait on the little boys first,' and, as I took no notice of the remark, seemed so determined his words should not be lost on me that he repeated them, with the addition, 'like you.' As before, it produced no immediate effect, but it afterward rose and rankled in my memory, and I was not able to keep clear of imagining vindictive things. In fact, to tell the truth, in both cases I felt that blood would have been sweet to me. . . . . My mode of thinking on these incidents no doubt had in it much of the character of insanity. . . . . The effect was that I got settled down into the fixed idea that contact with the thoughtless, evil world, in my state of body and mind, would impose upon me the necessity of committing crime in vindication of my honor. . . . . I let these bloody memories tinge my whole mind, and all its anticipations and resolutions for the future. . . . . 'I see,' I said to myself, in substance, 'that these galling collisions are the natural penalties of being imperfect.'

"It may be as well, for the prevention of misconceptions, to say that I never took one step toward putting any design thence arising into execution. I had no designs. I never armed myself, or, in fact, went any further than to rehearse the drama of revenge in my own mind. The pistol I bought was one which I would not have trusted for a moment to carry for the purpose of self-defense. . . . . Nevertheless, the events on the farm show that my wickedness was not altogether of a mimic kind, and I will not attempt to escape righteous judgment.

"I used to make many resolutions about regularity in habits of eating, which I found myself powerless to keep. A sense of depression and vacuity would come over me, aggravated by my solitary, monotonous life, I presume, and often by an obstructed state of the alimentary organs. . . . . It is a common feature in insanity or semi-insanity left to itself, I think. I also exerted my brain to the extent of abuse, I know, in the way of study. . . . I used to study Latin for a pastime, and often kept cudgeling my brains over Cicero and Cæsar until the top of my head was very sore. This solitary immersing of an enfeebled mind in study, with obliviousness to myself and all surroundings, was, no doubt, a help toward the grand consummation that took place in the fullness of things. . . . . I suffered a good deal from bodily ailments. My liver seemed to be thoroughly out of order and torpid. I had a feeling of hardness and inflammation in my sides regularly, a certain length of time after meals; digestion was bad, appetite irregular—in fact, every sign of a deadlock in the vital functions."

His mother and he removed to another village in 1871, when he was twenty-eight years of age, by which time there was but little question of his insanity, even among his relatives. I let him take the thread of the story again at this epoch:

"When my mother was making preparations for moving she asked me to help in packing up some chairs. I made an effort to apply myself to the task, but suddenly found myself overcome by my feelings, and before I knew what I was about I had shivered one of the chairs to fragments. A most unpromising omen! The fact is that I was, and had been for some time, in a state which any physician, knowing the facts, would have pronounced to be unmistakable insanity. But I had different ideas about what constituted insanity, and often thought to myself that if I did get put into an asylum, as had been threatened, they would not keep me, because they would see that I was perfectly rational. I have learned more about the subject since.

"Things of the kind I have told of had happened to me before, at uncertain intervals, during several years, an obstructed state of the bowels bringing on a turn. I would get into such a condition of exaggerated discomfort as to lose for a moment, or sometimes quite a spell, my control over my actions, and act very strangely. Sometimes I dashed down an article I happened to have in my hands, or demolished the first thing that came to hand; sometimes I gave vent to my feelings by grating my teeth, 'clawing' my face, and going through strange grimaces and agonizing contortions. My face seemed to me to be paralyzed when I had such turns, as if lifeless. The worst thing I ever did was when I flew at my mother in a sudden access of frenzy one day, when she had wrought upon my feelings by talking to me irritatingly, and bit out a mouthful of her hair. . . . When I was committed to the asylum, at a later day, it was reported as one of my symptoms that I had delusions about my mother being my enemy, etc., but nothing could be further from the truth. . . . I often grieved in secret over my inability to be a stay and protection to her, bereft as she was of all other support, but all in vain.

"In my new home I was in one of a row of houses, with strangers living near on both sides, and the sense of the presence of the evil which I had shrunk from so long weighed down upon me with crushing weight. After a while my spell of hypochondriacal despondency passed off, and I settled down into the way of living which I adhered to as long as I remained there. As to getting acquainted with my neighbors, or having any intercourse or dealings with them, that was altogether out of the question. . . . I now had more of the feeling of constraint, from the knowledge that I was moving under the eyes of people who were strangers to me, than the strangest of the strange could be to a person of the ordinary stamp. Sometimes I heard remarks which did not affect my feelings flatteringly, but that was not common.

"Along in June I had a worse spell than common of the kind of nervous stagnation or will-impotence of which I have spoken, and perpetrated some quite irregular acts before my fetters became slackened. In my despair I tore the collar from my shirt, tore the slippers I was wearing, dashed my fist into a tempting dish which my mother was offering me to eat, and other things of the kind. The house we occupied was owned by a maiden lady who lived with her sister in part of the house. . . . In the evening, after the other sister returned, who had been absent during the day, I overheard a few words which showed plainly enough that the events of the day were being discussed in no very gratified humor. It was evident that my acts were severely reprobated."

The next day the justice of the peace called upon him and admonished him to restrain himself, hinting of the asylum. Of this our author says:

"The dragon's tooth of reprimand that had been left in my mind grew into a monster, in whose presence I found it impossible to live, and I had a fresh access of despair. It was a hot June morning. I remember seizing a razor and flourishing it, and saying, 'Show me that rascal and I will slaughter him,' or words to that effect, meaning, of course, the justice of the peace."

Both homicidal and suicidal inclinations had long been haunting the secret corners of his mind, for three years before he tells of buying a pistol for the express purpose of making way with himself or some one else. On this day, after meeting the officer, he determined upon suicide. He walked out to two different country stores and bought ammunition. On his way back he passed some men in a field. They all looked at him, and one of them "laughed loud and mockingly, and then cried out, in a sort of squealing way, the intention of which could not be mistaken." Then he played a game of croquet with a young man at his uncle's, and overheard the young man make a covert and derisive remark. He continues:

"I passed the next day in brooding, silent melancholy. It was a rainy day and in accord with my feelings. . . . That night I wrote a little statement to be left behind. . . . It can not be said that I plunged thoughtlessly into the gulf of self-murder. I had from the first gaged the responsibility I was taking on myself, as fully as

> my mind was capable of doing it. I felt the whole weight of the condemnation that rested upon me for committing such a deed. . . . I passed some part of the hours of the night in sleep. In the morning my mother came to the door to see how I was, and I grasped her hand with a gesture of agonized despair. She took it as an indication that I was going to have one of my wild spells again, and, as she told me afterward, began to anticipate some work of demolition after I should come down-stairs. After she had gone down, I went and took the pistol from the stand-drawer, put on a fresh cap, got into bed again and propped up my head on the pillows, placed the muzzle of the pistol against the center of my forehead, and fired."

He lost considerable blood from the scalp-wound, but the bullet had glanced off; and, although he now tried to starve himself, he was up and about in a few days as usual, attending to his garden with bandaged forehead. He continues:

> "There were some steps taken toward getting me into an asylum after my abortive attempt at suicide, but as there were difficulties about it, and I appeared perfectly sensible and rational, my relatives concluded to let it rest.
>
> "From the time of my shooting until the next spring there was not much that deserves mention. How were my thoughts about suicide? It must be said that I had not totally renounced that idea. . . . I used very often to scan the beams in the wood-house and the coils of clothes-line in the garret. . . . The old difficulty of giving way under the slighting or displeasing demonstrations from others remained as bad as ever. I remember once I was so wrought upon by some trifling thing said or done by one of my relations that I kicked out the bottom of a cane-seat chair I was resting my feet on, in a sudden paroxysm of impotent emotion."

About this time he also made a futile attempt to poison himself by drinking a bottle of strong tincture of valerian that he had made himself. That incident he describes, and then proceeds:

> "It was my intention, when I began this sketch of my life, to give greatest prominence to that part beginning with my troubles in Clinton Street—that is to say, the period of confirmed lunacy with hallucinations, according to the world's avowed decision; but it appears at present that my project is not to go into fulfilment. I have been greatly delayed in doing as much as I have by lack of strength.
>
> "To make the account which I have given as full an exhibition of my condition at the time my hallucinations, if such, appeared, I will note some further defects in my mental action which I had noticed up to this time. First, two or three things indicating original lack of control over the brain by the will, or non-identification of my will with the action of my brain, and which I must count for predisposition. I have been troubled from my boyhood with a tendency of my brain to see things it ought not to see in what is placed before my eyes. This refractoriness does not extend to all kinds of monstrous visions, but is limited to the singling out of the lineaments of the human face in the outlines of objects seen. The annoyance I have experienced from this

has varied greatly, according to the state of my health. When I used to be sick with the fever and ague, I would lie in bed and gaze at the coarsely daubed window-shades in my bedroom, until I had made out every possible kind of a profile that could be distinguished.

"The other of the two most serious abnormal peculiarities is the supplying of missing articulations to vocal sounds, heard but not understood distinctly, so as to give my mind the impression of certain words, at the same time that I knew I had not understood. Sometimes I have been really cheated this way, and only found it out by inquiring afterward. This might not give conclusive proof of the deception, it is true. Not to violate privacies, I will illustrate supposititiously. If it were proclaimed aloud, far enough from me to allow the inflections but not the articulations to reach my ear with certainty—

WE SEE WHERE LIES THE DREADFUL SECRET!

my mind might involuntarily and instantaneously reshape it in such a way that I would understand:

DECEIVE WHERE LIES WERE EVER SACRED!

"My attention was always quite easily disturbed by noises, particularly talking. In boyhood the sound of voices in conversation at a little distance after I had retired to rest often gave me very serious annoyance, showing excessive irritability of the brain.

"Such was my mental state on the eve of my being overtaken by a more marvelously awful fate than ever fell to the lot of mortal man.

"My original purpose was to follow the incidents having a bearing on my mental fortunes with tolerable minuteness, in an unbroken chain, up to the time of reaching that wonderful state in which I have existed for the last six and one-half years.

"I shall be obliged to confine myself more to generalities.

"I was in such a towering state of morbid sensitiveness that a slight tinge of impertinence, brusqueness, or fancied contemptuousness in the manner of those I met, put me on the rack at once. . . . It began to occur to me after a little that my ears were becoming wonderfully acute for such things. Very often I would hear lively discussions on my character, and disputes about the proper epithets and titles to be applied to me, which I understood perfectly at an astonishing distance off. . . . I was wrought up to such a pitch that I formed a resolve that if I were given a sufficiently open provocation, I would attempt a bloody revenge, and on one occasion went out with a razor in my pocket. . . . I had an oppressive feeling of impotence, as if paralyzed, and suddenly did things I had no intention of doing, as in breaking glass. . . . I had a soreness all through my limbs which I compared to molten fire running through my nerves.

"I began to hear responses to and comments on my performances, and it gradually dawned upon me that I had been making myself a conspicuous object of curiosity to the whole neighborhood. . . . The comments heard grew more numerous and more and more derisive. . . . I had no suspicion at the time of any of the inspiration being drawn directly from my head. I do not say it was so. This is the debatable ground. . . . It was not until about a week later that it became evident to me that I was hearing my own thoughts given expression to by foreign wills and voices.

"I heard a great deal about 'inducting,' 'conducting,' 'sphere of influence,' sometimes even 'poles,' positive and negative, and my brain was constantly compared to a magnet. . . . I could find no better explanation myself for a long time than the theory of a fluid, similar to or the same as electricity, uniting brains.

"One was the story of an English physician who had become acquainted with my magnetic properties, and who was on the spot at the beginning, directing the experiment. He was stated to have been the first to form a perfect communication with the inducted brain, and he had drawn off my entire memory back to childhood, and had delivered it verbally in the presence of reporters from the city, who had taken it down. It was stated that the record was preserved in a number of thick volumes. These he had taken with him when he sailed for England during the most prosperous part of the experiment. It was further asserted that he continued in communication with my thoughts, and that wherever he went every one to whom he told the story of the new marvel was also set in connection with the magnetic current flowing from my head, and began to participate in my thoughts. . . . One word more of the English doctor. He is said to have declared that if he had assisted at my birth he would not have suffered me to remain alive, as the monstrous character of my organization could have been seen at a glance. . . . After the whole earth had become pervaded with the magnetism from my head, it would be felt as long as I lived, and the instant of my death would be thus signaled all over the globe, and would be noted and used by all nations as a new era from which to reckon time.

"I would think of the Bible, go and open it at haphazard, and just where my eye fell there was a passage that showed me *myself*. Once when I had been fretting about my ill success in getting my mother to accord with my views about my neighbors' doings, I hit upon this :

" 'And it shall come to pass that when any shall yet prophesy, then his father and his mother that begat him shall say unto him, Thou shalt not live, for thou speakest lies in the name of the Lord ; and his father and his mother that begat him shall thrust him through when he prophesieth', etc.—Zechariah, xiii.

"But the most perfect identity of all is to be found scattered through the Psalms" [of which he quotes several pages, and then continues] : "I do not intend to appropriate the spirit of these passages, or to make their language my own, but quote them thus collectively as an evidence of fact. I am myself but an inquirer. Do they express the experience of any certain person or persons? Or are they prophetic? . . . Can it be that the same thing that has happened to me has befallen another in ages long past, and that these are the traces of it?

"I have also found a most remarkably close application of many of the precepts and reflections of Thomas à Kempis in his 'Imitation of Christ.' He seems to keep the same character exhibited in the Psalms in view, only speaking as a monitor, instead of in his person. I presume I find myself mirrored in both these places, because I am an extreme case."

Gradually his delusions, burgeoning one from another, became so systematized that in the last year of his stay at this asylum he could write in his book :

> "The signs are too many and too evident to permit me to doubt that my destiny is bound up with the religion of the world. I steadfastly believe that the words in Jeremiah, 'Take forth the precious from the vile,' are addressed to me; and I can not be recreant to the holiest of duties. . . . I will not waste time in useless discussion, but start with the assumption that it is God's will that I should give the world my opinions.
>
> "If it comes to be generally believed that my sign is a fulfilment of Hebrew prophecy, I would recommend a transfer [of the Sabbath] to the day of the commandment. The very fact of a day one step removed being fixed on by both Christians and Mohammedans looks like an admission that another step remained to be taken.
>
> "Was it not the confidence of Jesus in the book spoken of above that made him say he knew the Father, when contending with believers in personified derangement?"

Quite a large part of the volume is devoted to expounding the Scriptures, in accordance with his delusion that he is a prophet come to reveal a new religion.

For instance, of Babel he says :

> "I find an application for the tower of Babel in my own insane history. I expect a confusion of the speech of the old sects to ensue likewise."

Of Abraham he remarks :

> "Abraham is accounted the father of all who believe in the Eternal. I believe I am chosen as his sign for the abolition of all dishonoring beliefs, as Abraham was set up against all idolators and pagans. . . . I have to note, in connection with the offering of Isaac by Abraham, that I find the date given as 1872 before Christ, coinciding with the year after Christ in which my ear-troubles commenced."

Of Esau :

> "We may take Esau for polytheistic religion, recognizing and deifying every force and passion that has dominion over the soul and destiny of man. . . . When it gave up its birthright for belief in a single judge, it pledged itself to go on and submit to be judged by the new master. I believe that the day of judgment has come."

Of the miracle of the rods :

> "The rods changed into serpents signify arguments becoming living convictions in the mind of Pharaoh. The evangelists' rods live as serpents in the minds of Christian believers, but I confidently expect that my rod will become a serpent that will swallow them all without trouble.
>
> "Israel is held responsible for the destruction of the heathen and their idols. I conceive that I am the Lord's instrument for the com-

pletion of this work, and that I have been shown these signs in the law that my hands might be strengthened.

"I can not shut my eyes to the fact that I have been made the world's sin-offering."

Of the prophets :

"The prophets I will take in a lump, with the assurance that no one can fail to see their connection with my destiny. There is a prophecy in Ezekiel, xxxiii, 30, which is very closely paralleled in my experience. . . . Jonah gives me a parable."

His discussions of theological questions are interesting, perfectly coherent and logical, although often fanciful. He pays tribute to the beautiful moral laws and righteousness of Christ, but is disposed to criticize His conduct as being inconsistent in one who claimed to partake of the omnipotence and omniscience of the Eternal. Of resurrection he says :

"If I conceive of a new body having the memory which I have of this body's life,—and I can find no other idea of the continuance of a soul's life except in the perpetuation or renewal of the memory,—would that in the new body be a *true* memory? Would it not be a hallucination? Would not that be an insane creation?"

In speaking of the years of his greatest mental aberration, he says :

"Here I come to more debatable territory, on which I and the rest of the world have until this present been at variance. I will, in deference to the other side, make use of the word *believe* in stating facts drawn from the region of my memory lying within this shadowy world. I will be permitted to say, therefore, that I *believe* that after settling down in the before-mentioned place, my brain was, by the gradual progress of events occurring naturally and according to the ordinary laws of human affairs, drawn into relations to the living actors around me, of an altogether unexampled kind—at all events, different from anything plainly recorded in the annals of past ages. I *believe* that the final result of such relations was the superinducing of a state of mental intercommunication through the medium of my sense of hearing.

"But this is a very old story, and merely a restatement of the perfectly well-known features of my alleged monomania. Let me pass on and give, as well as I am able, my own theory on which I explain these phenomena, which may have more interest. It is a question of personal identification. How does a man use his own brain? He can use it because it recognizes the actions of his members as belonging to the personal unit of which it forms the summit. Now the question is, can not a human brain under certain circumstances become so perverted as to recognize for itself, and without the volition of its bearer, the acts of other individuals as belonging to its life, as falling within its own memory? And if so, would not those individuals become partakers of the intellectuality of that brain, know its conceptions and ideas, while it thus recognized their motions, and become able to share its walks and ways? Such I believe to have been the result in myself, from the towering height of disintegration reached by my mental organism, by the gradual process which I have endeavored to faintly shadow forth in the preceding five chapters.

"Let us see whether it does not look probable that a mind in the habit of separating recognized observations from its own responsibility, considering them objectively, philosophizing on its own manner of working, driving the impotent and erratically acting part into a corner, as it were, would not be more exposed to such a fate as supposed than one acting unitedly, and right or wrong as a unit. It may not be susceptible of argument based on points of organic action, but it looks a plausible thing to me that the insane quality or element in such a brain might be acted on from without, and give itself up to such action, independent of the thinking will of that mind.

"But let us further suppose some little abnormality about the original constitution, a predisposition from a slightly dislocated arrangement of mind-apparatus and sense-apparatus.

"Such, say I once more, I believe to have been the case with myself, and such to be the true nature and essence of the things which have constituted my insanity. . . . I do not deny the fact of insanity, but I firmly believe that it is and has been, since the summer of 1872, an insanity involving the will, ideas, and acts of more than one individual.

"Notwithstanding my full and necessary faith in the reality of things as I have reasoned to prove them, I am still willing to concede that there has been more or less of purely subjective illusion mingled with these dual realities. Under one aspect the whole of this train of mental images and impressions which has whirled through my head has consisted of insane delusion. The effect on the state of my system has no doubt been analogous to that produced by delusions, and the nervous condition which preceded it was such as eventuates in the rise of delusions. Does not the development of delusions often have a compensating effect in freeing the nervous system in a manner from its trammels? Perhaps when this supervenes the brain becomes a chimney for the combustion of the matters which threatened to entirely interrupt the action of the system by clogging. The patient is then known as sensible on most subjects, but a confirmed monomaniac."

Certain peculiarities in his hallucinations possess considerable interest. They almost always referred to the intercommunication of brains. In July, 1878, he wrote out a list of specimen phrases which he had heard while sitting alone at an asylum window. Some of these I reproduce here:

"One thing you know, you know when you get your will in there you get him into a hell of misery."—"He ain't got any will there to fool away."—"Although you are knowing his ideas you connect with her will."—"Instead of connecting with his ideas you keep giving him to her."—"You can't get your will there till he connects his through to his thought."—"We are all the while trying to make him think himself."—"I think we ought to be making efforts to get the idea out on the hall."—"After they get the whole will he is in a hell of torture all the while."—"We keep hollering till we get him into a hell of horrors."—"You see, when there are two wills connected with the head at the same time, he ain't nowhere."

These were the voices of several men and women. In fact, his hal- ations were always polyphonic, and at times would be polyglot. did not address him directly, but spoke to one another about him. ldom had hallucinations of hearing except when the ear actually ved the sound of distant conversation or inarticulate noises; so that heir production it was usually necessary that there should be trans- on of vibrations to the auditory cortical area. As instances of the glot character of the voices on occasion, I relate the following:

Once he heard some one call out, "If he ain't a prophet there never a prophet—tabulas dedi ut vincerer." In tracing this Latin to its rce, he found it was a perversion of a phrase in a note to Whis- 's "Josephus": "Egomet tabulas detuli ut vincerer" (I myself carried the letter commanding that I be bound), attributed to Bellerophon, which he had once read.

At another time in a street-car, a German sitting next to him cried , "Das ist das grösste Mirakel von der ganzen Welt. Jeder lanke der ihm in den Kopf gekommen ist hat die ganze Village ört." (That is the greatest miracle in the world. The whole vil- e has heard every thought that has come into his head.) The gram- ical construction of the foreign phrases is open to criticism. The guage used by his invisible tormentors was always a peculiar dialect, n abounding in slang, which he considered the most hateful kind language, and which was such as he never voluntarily used in the composition of his own sentences. The hallucinations were usually boisterously satirical, teasing, quizzical, frequently accompanied by laughter.

Course and Prognosis.—The usual course of paranoia has just been outlined. Many cases, however, enter into a state of secondary dementia toward the last.

The prognosis is absolutely unfavorable. I do not know of a single case that has recovered. These patients may live to an advanced age, especially under the fostering care of an asylum. Remissions are occasionally noted.

Morbid Anatomy.—The disorder is purely functional. No pathological changes have been found in the brains of paranoiacs. In some instances asymmetrical arrangement of the convolutions has been noted. These belong in the category of stigmata of degeneration.

Treatment.—Therapy does little or nothing for the disease once it has become established. Sometimes complete change of environment brings about a remission. Constant physical occupation, hard work out-of-doors, is perhaps the most useful of remedial agents, in that by this means the mind is diverted from the constant contemplation of hallucinations and delusions, and through bodily fatigue is made to receive a considerable amount of repose. Labor acts as a counterirritant. By it episodic outbreaks of excitement may be aborted or reduced in intensity. Prevention naturally would be of vast importance, were one able to anticipate the coming catastrophe in the prodromal period. Children and youths who exhibit such symptoms as have been described as incident to the hypochondriacal epoch of the evolution of paranoia require a special system of education and training, in which occupation of the muscles and out-of-door life should play the chief rôle.

CHAPTER XV.

IDIOCY.

Definition.—In attempting to make a good definition and prepare a classification of idiocy, we meet with much the same difficulties as exist in connection with the allied subject of insanity. The innumerable definitions and classifications of insanity by different authorities are familiar to all students of morbid psychology. Each author feels called upon to be original in this particular, or at least to modify and improve upon the dicta of previous writers. This confusion is quite parallel in the matter of idiocy; and it is easy to understand why this should be so, for in both conditions we have deviations from the normal mental state of every possible shade and degree, depending upon a most varied pathology. The etiology is complex, and the psychic and somatic symptomatology multiform. There is no wonder, then, that the clinical picture is hard to draw, and the arrangement into clinical types difficult in the extreme. It is impossible to make any comparison between the psychological state of idiots and that of normal children, for the former is not only one in which the mental faculties are diversely undeveloped or impaired as regards their quantity, but there is infinite variation in the quality of the idiot's psychic functions. Likewise it is impossible to contrast the mental organization of the idiot with the intelligence of the lower animals, for the idiot is always abnormal, while the animal is a normal being in the zoölogical series to which he belongs.

What seems to be desirable in a definition is that there should be expressed in it the condition of mental weakness existing, the facts that the condition may be congenital or acquired, and may be due to a defect or some disease of the brain, and, further, that the condition is one belonging to the developmental period of life. A definition something like the following would seem to me to fairly express these desirable points:

Idiocy is mental feebleness due to disease or defect of the brain, congenital or acquired during its development.

Classification.—As regards classifications, they have been made upon a basis of symptomatology, psychology, etiology, craniology, teratology, and, to a certain extent, of pathology. But it seems to the writer that the time is not yet come for an accurately scientific classification of the forms of idiocy. It is much the best plan at present to adopt an artificial grouping, chiefly clinical, but pathological to the

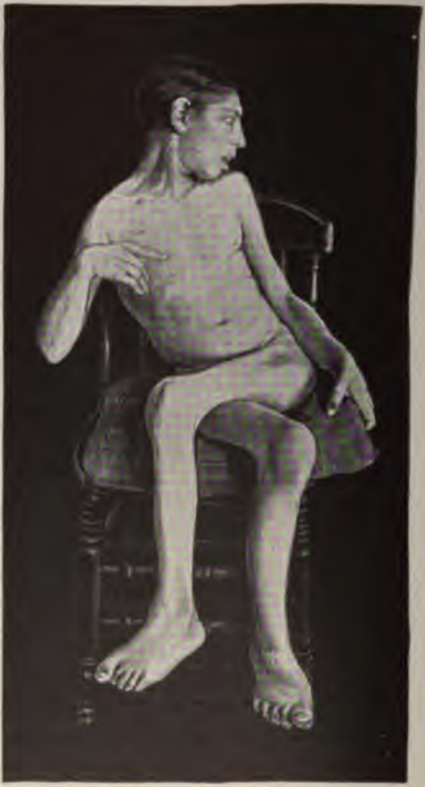

Fig. 315.—Diplegic idiot.

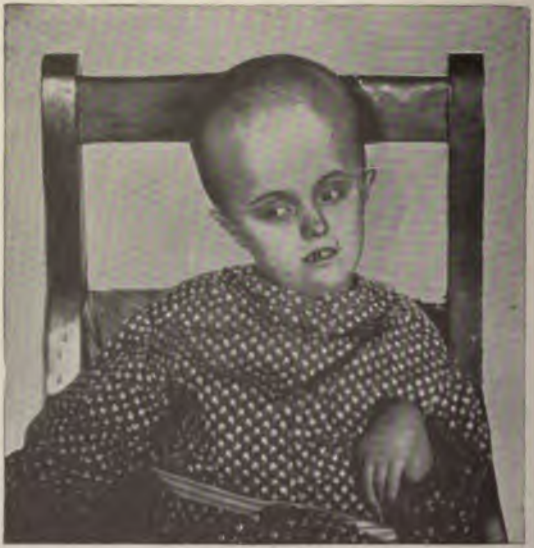

Fig. 316.—Extreme hydrocephalic idiocy, with diplegia.

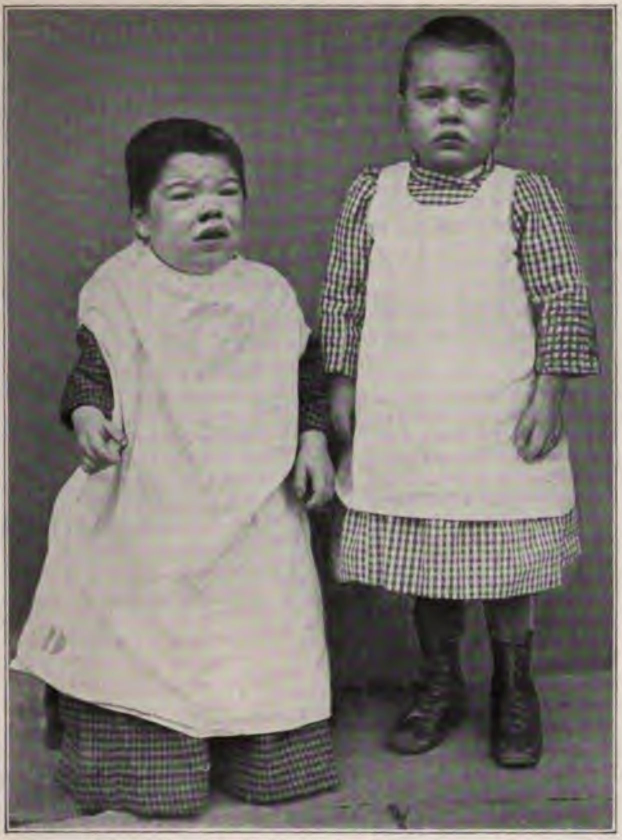

Fig. 317.—Cretin aged thirteen years standing beside normal brother aged four years (showing dwarfing of growth).

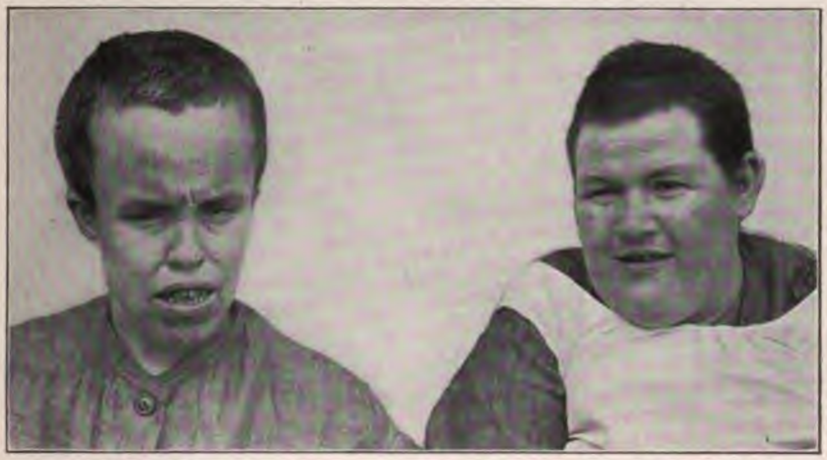

Fig. 318.—Hydrocephalic imbecile.

Fig. 319.—Idiot with multiple sclerosis.

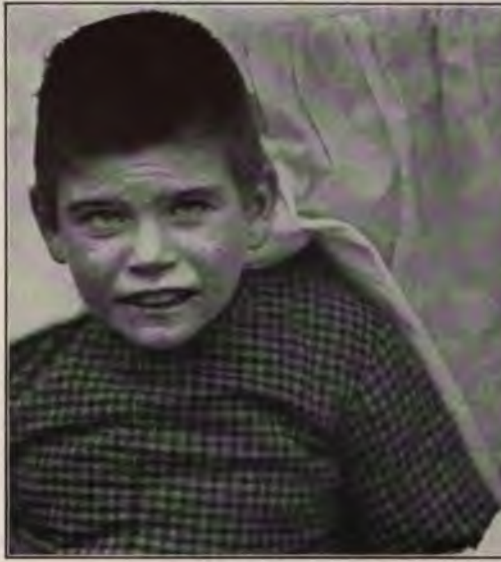

Fig. 320.—Microcephalic idiocy—wild, restless, quarrelsome, perverted.

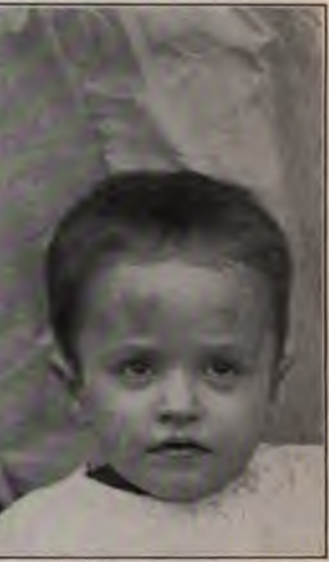

Fig. 321.—Hydrocephalic feeble-mindedness.

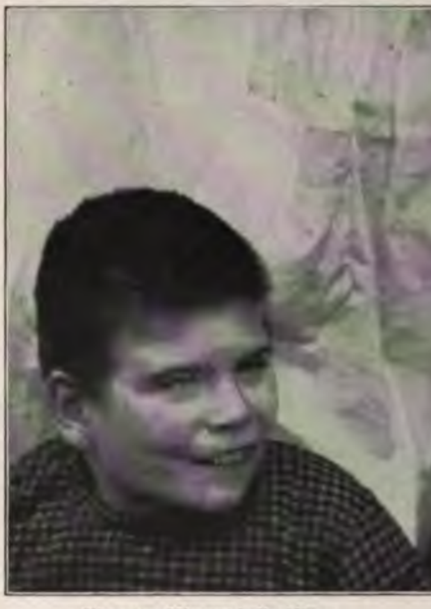

Fig. 322.—Microcephalic idiocy.

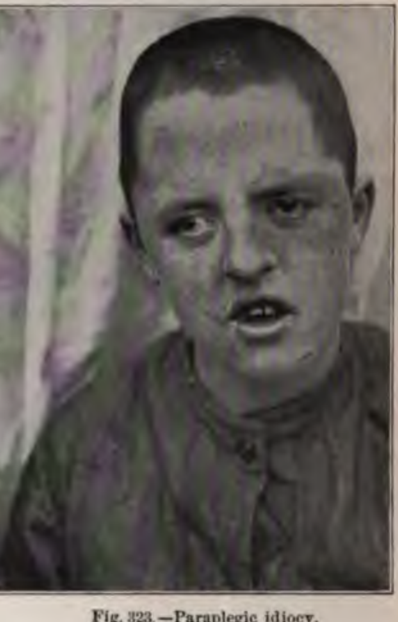

Fig. 323.—Paraplegic idiocy.

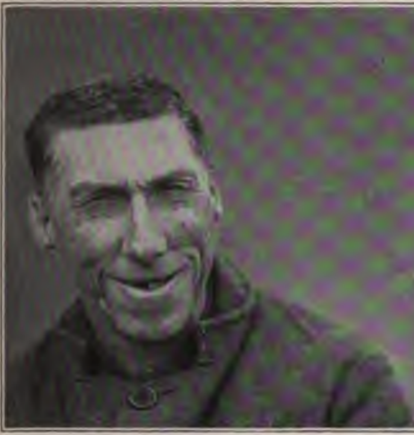

Fig. 324.—Microcephalic imbecile—good-natured and a fair worker.

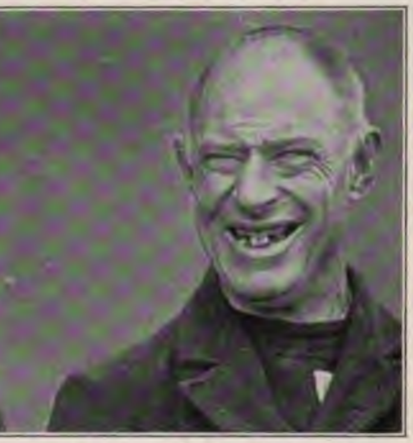

Fig. 325.—Good-natured imbecile—fair worker.

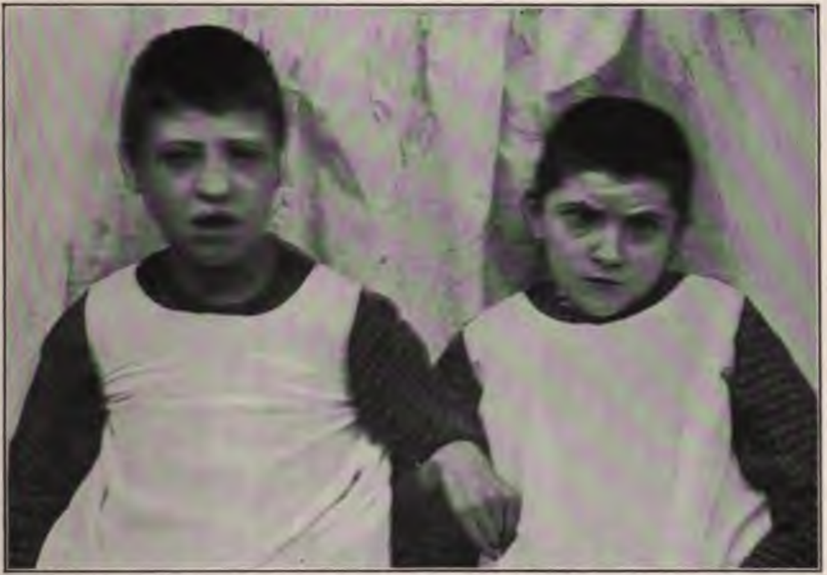

Fig. 326.—Two epileptic idiots.

extent of our latest knowledge. Almost any of the types of the divisions here made use of may be congenital or acquired. The term idiocy itself is generic, including as it does all degrees of mental impairment in early life. But the variations in degree or intensity of the mental weakness are indicated by the expressions: *idiocy*, for the lowest degree of mental disability; *imbecility*, for a higher degree, and *feeble-mindedness*, for the cases of idiocy in which the psychic faculties have their highest development. There is some confusion in literature as to the exact limitation and application of these degrees. Sollier has made an attempt to distinguish idiocy and imbecility, but his definition of imbecility is not tenable, in the opinion of the writer, for he describes a certain small class of imbeciles as representative of the whole order. It is to be remembered that in each of these degrees we have many gradations, and the entire series, from absolute idiocy to a normal state, leads up by progressive stages through various types of idiocy, several steps of imbecility, and numerous shades of feeble-mindedness, until the borderland between the highest degenerate and the normal individual is almost indefinable.

The highest group includes a rather well-defined class of feeble-minded: the "backward children," the *enfants arrièrés* of the French, the *tardivi* of the Italians, and the *Geistig-zurückgebliebene* of the Germans. The difficulty is not so much in the delimitation of this class, as in the separation of the group of idiots and imbeciles. It is easy to make the classification on seeing the cases, but to convey to others the differentiation by description is far from being so, because of the many features—physical, motor, and mental—which are concerned in such division. The writer, while employing the term idiocy often to include all of these degrees, would define the idiot proper as an individual able to give little or no care to his person; incapable of intelligent communication, barely able to express his material wants, most awkward and ungainly in his movements, if he move at all, and presenting marked evidence in his lack of expression, apathetic attitudes, and physical stigmata of degeneration, of the profound stunting of his mental and physical development. On the other hand, the imbecile is able to care for his person and dress, attend to his physical wants, comprehend fairly what is said to him, carry out orders more or less intelligently, is often able to speak well (though sometimes speech may be impossible to a very intelligent imbecile); if not paralyzed, he has good use of all his muscles; he is not destitute of expression, though the expression may vary from an evil, mischievous, cunning cast of countenance to one of rollicking good nature; there are fewer stigmata of degeneration in this class than among idiots.

The clinicopathological grouping of the varieties of idiocy which the writer has found most useful to him in his work at the Randall's Island Hospital for Idiots is as follows:

1. Hydrocephalic idiocy.
2. Microcephalic idiocy.
3. Paralytic idiocy.
4. Epileptic idiocy.

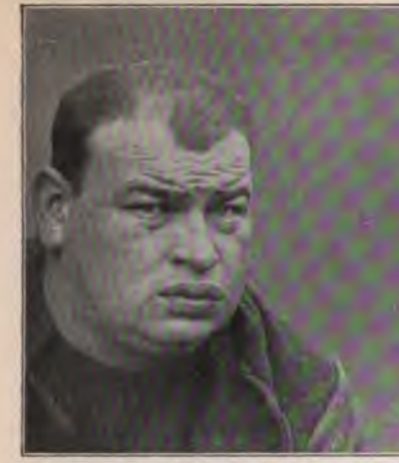

Fig. 327.—Epileptic idiocy.

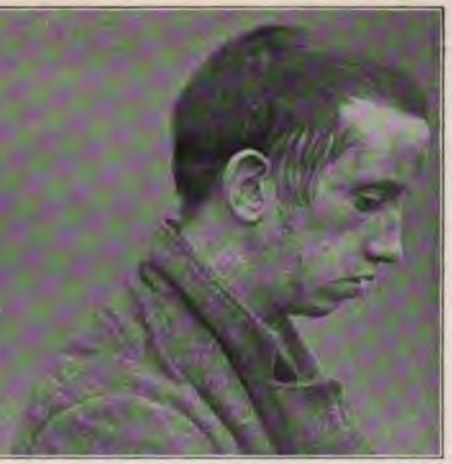

Fig. 328.—Imbecile, with extreme dolichocephaly. (Length-breadth index, 51.)

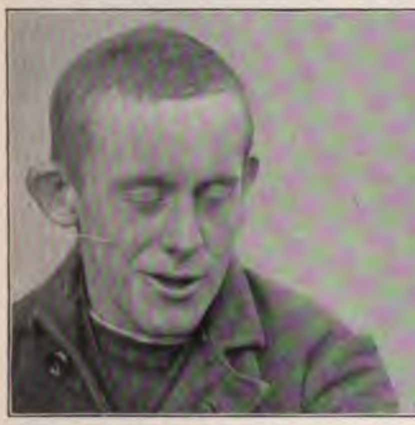

Fig. 329.—Hemiplegic idiocy. (Blainville ears.)

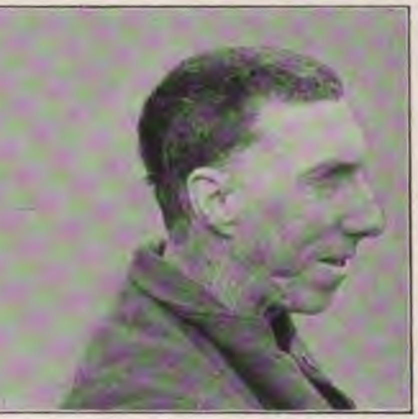

Fig. 330.—Microcephalic imbecile.

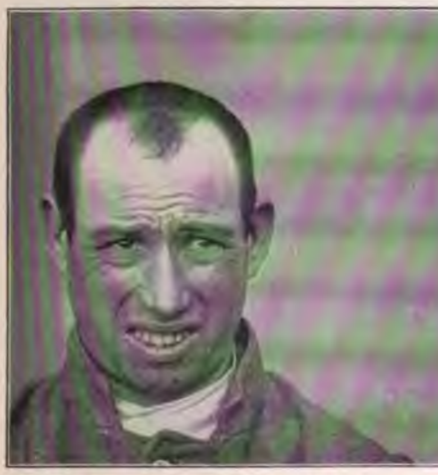

Fig. 331.—Idiocy as a result of dementia from acute insanity in childhood.

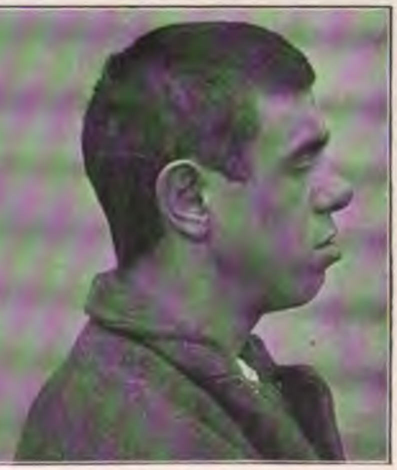

Fig. 332.—Epileptic idiocy.

5. Traumatic idiocy.
6. Sensorial idiocy.
7. Meningitic idiocy.
8. Myxedematous idiocy, or cretinism.
9. Amaurotic idiocy.
10. Idiots savants.

It is impossible, in the brief space allotted this subject, to discuss these various forms of idiocy in detail. The reader must be referred to special works and articles on idiocy—to the general works of Downs, Shuttleworth, Voisin, Sollier, etc.—and to the monographs by the writer and others. Hydrocephalic, microcephalic, paralytic, epileptic, and traumatic idiocy are readily recognized by their symptoms or history. Sensorial idiocy is a form due to the congenital or early loss of two such senses as sight and hearing. Properly treated, these patients are capable of normal mental development (Helen Kellar and Laura Bridgman). Meningitic idiocy can usually be diagnosticated only by autopsy, unless the history or exacerbations in the course of the disease demonstrate its origin. Cretinism is a rare form which has been fully described in many brochures in recent years. The amaurotic form is still rarer. There are only two of these in the Randall's Island Asylum among many hundreds of idiots.

The term *idiots savants* is applied to all such idiots, imbeciles, or feeble-minded as exhibit special aptitudes of one kind or another, always out of proportion to their intellectual development in other directions, and often remarkable as compared with similar accomplishments or faculties in normal individuals.

There are many cases of the kind recorded in literature, and it is not at all uncommon to hear of idiots in our newspapers and museums who are exhibited as musical prodigies, "calculating boys," and the like. Beyond the fact of the existence of such curiosities, and the record of their deeds, there has been little or nothing written in explanation of these phenomena. The psychology of the condition is exceedingly obscure; and even were the psychological processes which underlie special aptitudes understood, there would still remain the mystery of the manifestation of particular talents or faculties in minds otherwise blank or defective.

The aptitudes may be summarized as follows:

Arithmetical faculty, musical faculty, special memories, imitative faculty, modeling faculty, delineative faculty, faculty for painting, aptitude for games (draughts, etc.), aptitude for buffoonery. (See article by author on "*Idiots Savants*" in Appleton's "Popular Science Monthly," December, 1896, in which a history of some remarkable examples is given. See also page 869 of this book.)

General Etiology.—There are nearly twice as many male as female idiots. In idiocy due to prolonged or difficult labor, this disproportion is even larger (three males to one female)—a fact to be explained probably by the larger size of the male infant. The causes of idiocy may be classified as follows:

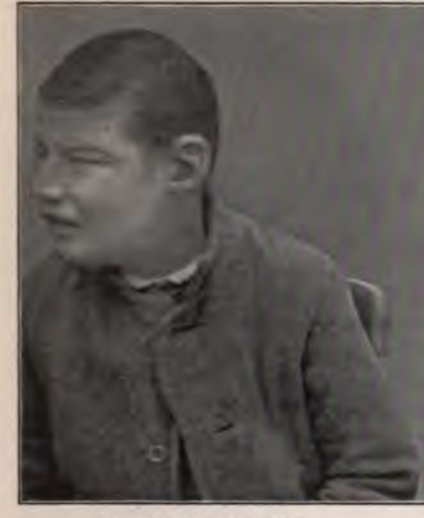

Fig. 333.—Paraplegic idiocy.

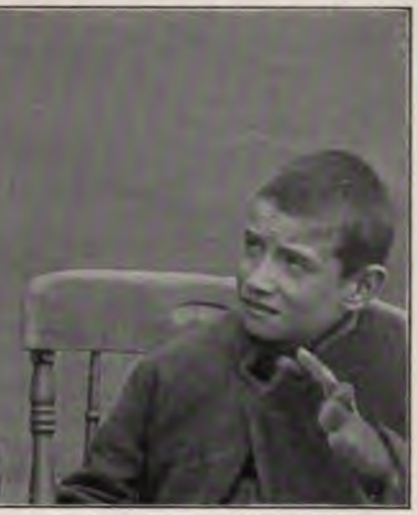

Fig. 334.—Idiocy after acute insanity of childhood. Peculiar tic of fingers.

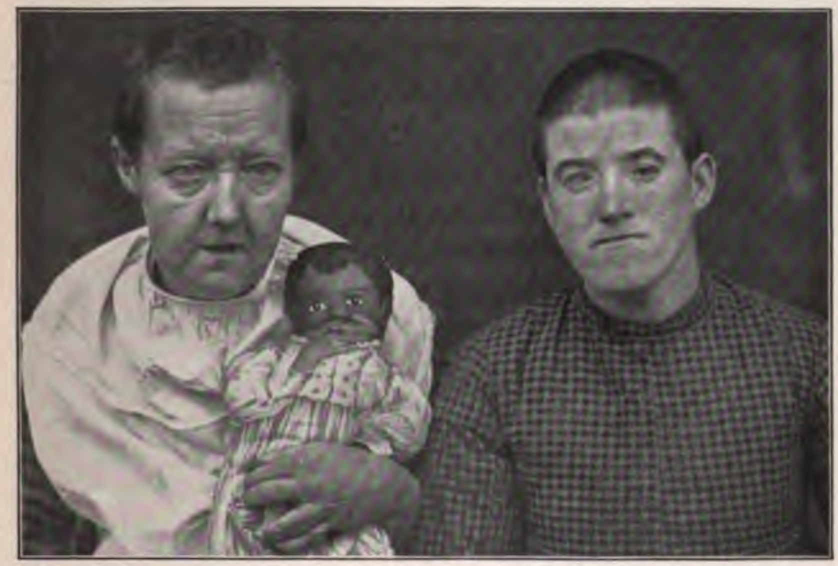

Fig. 335.—Two imbeciles, one epileptic and one of unknown origin (both homosexual perverts).

Degenerative			Hereditary transformation of nervous and mental diseases. Pathological heredity in the form of vitiating diseases or habits (tuberculosis, rheumatism, gout, herpetism, syphilis, alcoholism, etc.). Sociological factors (extreme youth of parents, extreme age of parents, disproportionate age of parents, consanguinity).
Adventitious	Gestational	Maternal	Trauma, shock, fright, diseases, maternal impressions.
		Fetal Disorders	Syphilis, heart disease, arteritis, morbid processes in the brain and meninges, twin pregnancy.
	Parturitional		Difficult labor, primogeniture, premature birth, asphyxia at birth, instrumental injuries, pressure on the cord.
	Postnatal		Convulsions, cerebral diseases, trauma to the head, febrile diseases, mental shock, sunstroke, overpressure at school.

The relations of heredity to idiocy are much the same as those of heredity to the psychoses described in the chapter on General Etiology of Insanity. The statistics available (such as those of Shuttleworth and Beach, Langdon Down, Kerlin, and Piper) seem to show neurotic inheritance in about forty to fifty per cent. of idiots. The stigmata of degeneration, which are so marked in idiocy, are described in an earlier chapter. As regards tuberculosis and scrofula in the parents, the percentages of these authors vary from fifteen to thirty per cent. Alcoholism takes a high place among the causes of progressive hereditary degeneration (nine to sixteen per cent.). The writer has found that hereditary syphilis is a comparatively rarer cause of idiocy than many would suppose, and this is supported by the statistics of the authors named above (one to two per cent.). As regards consanguinity, the statistics show that the proportion of idiotic offspring of cousins to the number of idiots is very slightly in excess of the number of consanguineous marriages to marriages in general.

Gestational causes vary, according to the statistics, from eleven to thirty per cent. Parturitional factors (meningeal hemorrhage from prolonged labor, asphyxia at birth, premature birth, pressure on the cord, forceps injuries, etc.) are active in about eighteen per cent. It may be said that forceps injuries are far less dangerous to the child than tedious labor. Among adventitious causes infantile convulsions occupy a preëminent position (over 25 per cent.). But we must remember that the convulsions may act as a real cause, by inducing meningeal hemorrhage; or convulsions may be merely an associated symptom of a meningeal hemorrhage or other pathological condition due to some other common factor. Cerebral diseases (meningitis, hydrocephalus, hemorrhage, thrombosis, embolism, tumor, and abscess) follow infantile convulsions in the statistical table of causes (eight to nine per cent.).

Acute febrile diseases induce idiocy in some six per cent. of cases.

These diseases are scarlet fever, measles, whooping-cough, typhoid fever, small-pox, and diphtheria. How they act is not yet known. Sometimes it is through meningeal hemorrhage induced by the convulsions so common at the onset or during the course of these maladies. Possibly at other times it is through an infectious encephalitis, or microbic embolism or thrombosis. Trauma to the head, mental shock, sunstroke, and "cramming" at school have a small, yet appreciable, share in the production of idiocy (probably two to five per cent. altogether). The author has found, in his own experience, that insanity in children is an occasional cause of idiocy. In the adult such mental enfeeblement after insanity is a secondary dementia, but in the growing child this secondary dementia is preferably termed idiocy.

General Symptomatology.—Since idiocy, as well as its varying degrees of imbecility and feeble-mindedness, depends upon some sort of congenital or acquired defect or disease of the brain interfering with its normal evolution, it is clear that the cerebral functions may be all of them more or less involved, and that no particular psychic faculty can be selected as the one whose disorder retards or influences the development of the other faculties. Seguin is, therefore, hardly correct in stating that the condition of the mental faculties in idiots is normal, though diminished, and that merely the will is lacking to give them proper direction. Sollier has given us one of the best and latest studies of the psychology of idiocy.[1] Following Ribot and others, he maintains that the slow development of the cerebral faculties is due to want of attention; that spontaneous attention is caused by affective states brought into action by sensations, and that those young children are the most attentive whose nervous systems are most easily stimulated. Hence the faculty of attention is closely related to the activity of the sensations. The greater the power of attention, the more intelligent does the individual become. In idiocy, owing to the diminution or loss of the power of attention, the perceptions aroused by sensations are more or less indefinite, and the resultant idea likewise ill-defined. Sensations become more numerous as the organism develops, and the lack of ideas and recognitions becomes more noticeable.

Following somewhat the natural order of such examination, with the excellent work of Sollier[2] as a guide, we first take up the senses, those avenues which lead to psychological development.

Sight.—Between seven and eight per cent. of idiots are congenitally blind. It is necessary to determine whether the blindness is due to defect of the visual apparatus or to lack of attention. Blindness does not preclude the possibility of education, for some idiots with defect of this sense may be educated to a moderate degree. When idiots can look, without in reality seeing, the apparent blindness is due to a complete absence of attention. In idiots less affected, a greater variety of objects will attract their attention. In the higher grades of idiocy (imbecility and feeble-mindedness) vision may be as good as in normal man. But many present certain visual and ocular defects, such as hypermetropia, defective color-vision, strabismus, nystagmus, congenital cataract, inequality of the pupils, microphthalmos, and the like. In hemiplegic idiocy or imbecility we may find hemianopia. But the determination of the acuity of vision is difficult in this class of individuals. The perception of different colors is often possible in the milder degrees of idiocy. Good binocular vision is uncommon in idiots. The normal child takes pleasure in the sight of objects as early as the eleventh day, the eyes are normally coördinated by the end of the second month, and he begins to distinguish colors correctly at about the age of two years.

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[1] "Psychologie de l'idiot et de l'imbecile," Paris, 1891.

[2] The author, while differing from Sollier materially in some of his conclusions, is indebted to his work for many of the details of the psychological symptoms of idiocy.

Hearing.—There is a somewhat analogous condition of the organs of hearing. It is not always easy to determine whether an idiot is deaf from defect in the auditory apparatus or only sensorially deaf. Idiocy of mild degree is not infrequently induced by deprivation of this sense. In the higher grades of idiocy hearing is nearly always normal. Deaf-mutism can not be considered as common. The normal child hears on the fourth day, and is pleased with music in the second month.

Taste.—This sense is frequently affected. Gluttony is a marked feature in idiocy. It is common for idiots to eat without mastication; many present a precocious taste for alcohol. This is especially true of the higher grades. A difficulty in distinguishing the simple tastes (salt, sweet, bitter, and sour) is not infrequently met with in the milder types, as well as in those with great mental impairment. Inversions and perversions of taste are observed. The normal child evinces a sensibility to taste at the end of the first week.

Smell.—In the normal child strong-smelling substances produce mimetic movements on the day of birth. In idiocy the sense may be much impaired, perverted, or absent.

Tactile Pain and Muscular Sensibility.—As a rule, sensibility to touch and pain is uniformly diminished in idiocy of all degrees, mostly through lack of attention. There may be complete anesthesia and analgesia, particularly in the lower grades. On the other hand, there are cases in which the sense of touch may be educated to a high degree of delicacy. It is almost impossible to study the muscular sense in idiots, but it is apt to be impaired in proportion to the other senses. The normal child starts at gentle touches on the second and third days, and manifests muscular sense as early as the eighth week.

Thermic Sensibility.—What has been said of touch and pain applies likewise to the temperature sense. But their vasomotor systems are susceptible to the influences of cold and exposure, and their resistance to external influences and diseases is such that many of them die of pulmonary affections. Some become more stupid in cold weather and brighter in warm weather, while an elevation of bodily temperature (fever) is accompanied by evidences of more active cerebration.

Morbid Movements.—A small number of idiots exhibit no motility at all, but remain perfectly inert. But the majority are apt to be in constant motion. These movements tend to take on a rhythmic and automatic character. I do not here refer to such morbid movements as epilepsy, athetosis, associated movements, ataxia, and chorea, often present in paralytic idiocy; nor to tremor, found in sclerotic cases; but to a group of automatic or impulsive movements.

These forms of movements are among the most common and striking symptoms immediately noticed in going through an institution for idiots. A very large proportion of the inmates are observed to be in continual motion. As a rule, the most frequent rhythmic movement is an anteroposterior oscillation. The patient, in a sitting attitude, sways his body slowly or rapidly backward and forward. Sometimes the oscillation is from side to side. Occasionally the hands and fingers are rapidly or slowly flexed and extended, and brought up to the face in movements similar to those in athetosis, but differing from them in that they are entirely subject to the will, just as are the oscillations alluded to. Walking to and fro, rotating, dancing, and so on, are more elaborate forms of the same kind of impulsive movement. Similar movements occur in the insane, as is well known, and particularly in conditions of greatly enfeebled mind, such as secondary dementia. They are spontaneous movements, seeming to have no relation to any stimulation of the brain giving rise to a motor expression. Generally the movements cease for a time when any sensory impression, such as the appearance of a stranger in the room or being spoken to, temporarily alters the feeble current of thought or excites the mental blankness which has given rise to the automatic movement. Children and young animals are full of spontaneous movements, undoubtedly due to impressions received at some time during their lives, or, it may be, impressions inherited; and, while these spontaneous movements of children are undoubtedly similar in their nature to the automatic movements of dements and idiots just described, they do not often present the rhythmic character of the latter. It is probable that in the feeble mind, upon which nerve stimuli seldom make an impression, the simple old motor expressions are retained, repeated, and become habitual or automatic. Automatism of movement is thus a sign of little aptitude or impressionability, so far as fresh mental stimulation is concerned. In the idiot the impulsive rhythmic movements just described may be regarded as the habitual motor expression of the simplest and oldest stimuli; whereas, in the secondary dement, the analogous automatic movements are to be looked upon as reversions to the spontaneous movements of infancy. The smiles and grimaces of idiots and imbeciles belong to the same category of infantile spontaneous motor expressions.

There is probably a certain amount of pleasure in the movements in many cases, as sometimes they manifest displeasure if prevented from executing them. There is nearly always a difficulty out of proportion to the intellectual development for idiots to perform associated movements with a definite object. They may be able to talk and read, and even write, yet be unable to dress themselves. This is often a fault remediable by education, according to Seguin.

Right-handedness and Left-handedness.—Some twelve per cent. of all children, idiot and normal, are left-handed; but while eighty-eight per cent. of normal children are right-handed, only seventy-two per cent. of idiots use their right hand in preference, the remaining sixteen per cent. being ambidextrous. This peculiarity is said to be present also among criminals.

Voluntary Movements.—Many idiots do not learn to walk at all, either because of general debility, inability to learn, or paralysis. In such as do acquire the ability to walk there is great retardation in its acquisition. This is also true of other uses of the voluntary muscles for the common acts of daily life, such as carrying food to the mouth, and so on. They are either never learned or they are acquired late.

Organic Sensations.—The keenness of visceral sensibility is more or less diminished in all idiots. The sensations of hunger and thirst are lessened, though only very rarely absent. The feeling of satiety after a hearty meal is seldom felt by them; so that if left to themselves, they would eat on indefinitely. The necessity of defecation and micturition is not perceived at all by profound idiots. In the lower and middle grades of idiocy it is often difficult to diagnosticate visceral disease, owing to the bluntness of somatic sensations, and they may die without giving any appreciable symptoms. This masking of disease in idiocy is quite analogous to the masking of disease in various insanities. The feeble-minded and imbeciles not infrequently mislead the physician by exaggeration, concealment, or falsehood.

Attention.—The lack of the faculty of attention is one of the chief characteristics of idiocy. Naturally, it varies in degree from complete nullity to a simple diminution of the faculty, but it is always lessened. The fundamental elements of the faculty are deficient. These fundamental elements are: The integrity of sensory impressions delivered to the brain; an emotional state of pleasure, pain, or interest in such sensations; motor expressions in the eyes, face, limbs, or body of the impressions received. There are two forms of attention, according to Ribot and Sollier, one of which is natural or spontaneous, and the other voluntary, established by education. The latter can not exist without the former.

There are two qualities in attention that are of importance—viz., intensity and duration.

Thus, attention is impaired in idiocy by the defective senses, which convey to the brain feeble impressions. The second element, the affective state, is notably lacking in idiots. The motor factor of attention is deranged in idiocy in a great variety of ways (general weakness, paralysis, contracture, epilepsy, chorea, ataxia, automatic and impulsive movements, and the like). The intensity and duration of attention are restricted to the last degree in this class of individuals.

The intelligence and the possibility of education depend directly upon the power of the faculty of both spontaneous and voluntary attention. It is probable that the faculty is localized chiefly in the frontal lobes of the brain. Ferrier considers it proportionate to the development of these lobes, and some very convincing experiments recently published by Bianchi make it quite certain that the frontal lobes are the seat of this faculty. In idiots great lack of attention is coincident with diminutive size of the frontal lobes.

In the low grades of idiocy spontaneous attention is almost null, and education is impossible. The higher the degree of idiocy, the greater the degree of spontaneous attention presented, which may be so appealed to as to develop it into voluntary attention, with intellectual progress as a consequence. With idiots, as with the lower animals, attention is always connected with the sense most perfectly developed, which, in the former, is that of sight. The attention of idiots is most easily aroused through the eyes. Exercises of the attention may thus be employed in the diagnosis of states of intellectual weakness. We find idiots without attention absolutely ineducable, leading a vegetative existence; others, again, exhibiting both spontaneous and voluntary attention, but in flashes, as it were, of brief duration and faint in nature; and still others more or less capable of prolonged and habitual attention. It is only in the last-named group of individuals that education is to any considerable degree feasible. The education appeals in some to the simplest sentiments only (such as curiosity, selfishness, the desire of reward), in others attention is aroused by appeals to a higher affective order (such as interest, ambition, and emulation), and in still others attention may be aroused and sustained by habit.

Since the power of attention directed to external events is so feebly developed in idiots, it is not surprising that attention to internal happenings, or reflection, should be totally absent in all grades of idiocy.

Ribot regards voluntary attention as habitual and disciplined spontaneous attention, as an adaptation to the conditions of a higher social life, as a sociological phenomenon. When the development of voluntary attention is rudimentary, and the resulting intellectual defect is marked, as in the lower grades of idiocy, there are no serious consequences from the sociological point of view. Sollier calls the idiot *extra*-social, and makes the imbecile quite distinct as *anti*-social, claiming that in the latter there is an undefined amount of voluntary attention, combined with a relative, though perverted, intelligence, which two factors render him often a dangerous member of society. He speaks of the instability of the attention of the imbecile. At one moment it may be faint, at another intense as in normal man. He passes from one subject to another with the greatest ease, a characteristic which may even be observed in his infancy. Serious matters must be continually repeated to him to make him understand. He grasps the first part of a sentence, and forms his ideas from that, without waiting for the sentence to be completed. He frequently interrupts, and there is no time to answer one question before another is put. Sollier further goes on to say that this instability of the attention for external objects or ideas is seen also in the acts of the imbecile, who is incapable of intelligent labor, and accomplishes his tasks, when uniform, by a certain kind of automatism, without due appreciation of the object of his work. When the object is understood, the imbecile believes he can attain it immediately, and, seeing the first step only, is prevented by failure of attention from properly completing the work or doing it at all. He seems to forget that he has begun, and as a consequence, unless watched, may spoil whatever he attempts. Other imbeciles refuse to work, but make themselves very busy and important in watching and supervising the occupations of others.

Sollier calls them vagabonds. They wander away not knowing where, marching straight before them, with indifference to the welfare of the friends or relatives they desert; traveling by night and hiding by day; undisciplined, indolent, and mischievous.

This attempt to separate idiots and imbeciles into two distinct classes of extrasocial and antisocial is, to my mind, not justifiable. Sollier has here described a certain class of imbeciles only, and the description is very true to nature, but it is only a group which does not merit an especial classification. As regards attention, we should still hold to the terms idiocy, imbecility, and feeble-mindedness, as representing degrees of lack of attention, from complete or almost complete absence to mere diminution of the faculty. The adult imbecile, in the middle grade, would have the varying and imperfect attention of a backward child, and his ideas, speech, and conduct would vary with his temperament, with his docility or perversity; in short, with the innate differences of character and individuality, which may be as manifest in imbeciles as in normal children. Imbeciles may and do become vagabonds, uncertain, mischievous, indolent, antisocial; but they may, on the other hand, be good-natured, trusty, docile, industrious. Many of them, too, may show special aptitudes in certain directions. As to education, the difficulties are that in some it is hard to attract the attention, and in others to maintain it.

Reflection.—The internal form of attention (reflection of Ribot), in which images and ideas constitute the subject-matter, is quite deficient in the lower grades of idiocy, but is present in imbecility and feeble-mindedness in varying degrees. It is never perfectly developed, as in normal man.

Preoccupation.—This is absent in profound idiocy and feeble in the higher grades. A small proportion of imbeciles are capable of preoccupation, but of an indefinite nature, and sometimes taking on the character of a fixed idea. Often their interest is not aroused so much by what benefits and interests mankind in general as by bad actions, criminal or egoistic sentiments that attract their attention and arouse reflection and preoccupation which may result in felony or crime. Many are too selfish to care for the troubles of others, and too stupid to have preoccupations purely intellectual.

Instincts.—The instincts in idiocy are generally defective. The defect may be imperfection of development or an actual derangement or perversion. The instinct of hunger is present in almost all grades of idiocy, and is so little inhibited that it is often pushed to the extent of gluttony. The instinct of self-preservation is impaired in nearly all, absent in profound idiocy, ungoverned by proper judgment in the milder forms. In some there is no sense of fear, and self-injury is possible. In others there is a comprehension of danger and an avoidance of it, or possibly an overweening egoism which may lead to a belief in their power to overcome it. Suicide occurs in imbeciles and feeble-minded, sometimes without determinable cause, sometimes as a result of morbid impulse.

Sleep is good among all classes of idiots, while in the lower grades it may be both profound and excessive. Whether they dream or not depends solely upon the degree of mental development.

The desire and need of voluntary muscular movement varies with the scale of intelligence, being absent in the profounder degrees of idiocy, and approximating the normal the higher the psychic development. The automatic and impulsive movements in some may represent a fulfilment of the normal need, and the extreme restlessness of others is surely a perversion of the natural desire.

The sexual instinct may be absent, impaired, exaggerated, or perverted. It is seldom normal. Idiots of all degrees present many degenerative stigmata as regards the genital organs, more numerous in direct proportion to the mental impairment. Among these anomalies are: cryptorchismus, unilateral or bilateral microrchidia, spurious hermaphroditism, insufficient development of the entire genital apparatus, hypospadias or epispadias; defect, torsion, or great volume of the prepuce; median fissure of the scrotum, imperforate meatus, abnormally large or small labia, excessive development of the clitoris, hypertrophied labia minora, pigmentation of the labia minora, imperforate vulva, atresia of or double vagina, and uterus bicornis. Puberty is often retarded, but occasionally is early; often it is normal. Masturbation is exceedingly common among all classes of idiots of both sexes. In the profound degrees it is automatic; in the higher it is purposive. Onanism *à deux* and sodomy are frequently discovered among imbeciles and feeble-minded, and sexual psychopathies of the most shocking nature are not uncommonly manifested in some because of the combination of the strong sexual instinct and absence of moral sensibility.

The instinct of imitation, which is a low form of instinct, and strong in children and many lower animals, is one to which idiots are very susceptible. It is usually a purely instinctive or passive imitation, seldom an intellectual or active imitation. Its intensity depends much, however, upon the scale of intelligence to which the idiot rises. It is very apt to be shown in the form which is concerned with moral contagion; so that the acts and language of the vicious, mischievous, coarse, and vulgar are most willingly imitated. Simulation is very common among the more intelligent classes of idiots.

Special Aptitudes.—In the so-called idiots savants we note the development of special aptitudes, occasionally remarkable, more often only noteworthy in contrast to the general mental vacuity. These aptitudes are usually in the direction of music, mathematics, the mechanical arts, building, wood-carving, drawing, painting, memory for facts or dates, playing games, and of a low order of wit or drollery. The occasional preëminence of some particular faculty, where all other traits are defective, would almost lead one to believe in a heterotopia of gray matter in some special locality. Music, the most sensual of the arts, seems to appeal especially to this class of individuals. Often the rhythm of it seems to influence the rhythm of their automatic movements, or it soothes their restlessness or stops their cries. Sometimes unteachable idiots are able to retain, recall, and hum a moderately difficult tune, while higher grades may learn to play instruments by ear, though not by note. Next to aptitude for music, that for mental arithmetic is often surprising. There are also occasional instances of the other talents just mentioned, and doubtless the court fools of the past, with their mischievous pranks and quaint remarks, were recruited to a great extent from the imbecile class.

Play.—There is a lack in all classes of idiots, and in direct proportion to the degree of mental defect, of that " superfluous activity which is expended in the form of play." The activity and attention of normal children are mainly developed through play. This avenue of education is, unfortunately to a considerable degree, closed in idiocy. The lower grades, if they manifest a tendency to play at all, do so in a rudimentary and solitary way, and in adolescence still cling to the simple games of infancy. With others, higher in the scale of intelligence, there is still defect of the play instinct, and a proclivity often to prefer games in which noisiness, destructiveness, and other evidence of rather brutal traits are paramount. Sometimes these games are carried on good-naturedly ; at others, selfishness, irritability, quarrelsomeness, and a more or less ungovernable nature are evinced.

Civility and politeness may be taught to many, but naturally with difficulty to the lower grades and to such individuals of the higher as are hard to train in other directions, because of innate vices of temperament and character.

Destructiveness, a propensity even in normal children at an early age, is an especial attribute of all classes of idiots. In those of low degree it is automatic and possibly a rudimentary form of superfluous activity (play), but in some individuals of the superior grades there seems, at times, to be a vicious satisfaction in inflicting damage or injury, which may even lead to the manifestation of homicidal proclivities or a tendency to arson (pyromania). Self-mutilation or injury may be a result of the love of destruction in the profounder degrees of idiocy.

Sentiments.—In the lowest forms of idiocy the sentiments and sensations are rudimentary, or may be altogether absent. As a rule, one may discover various degrees of pleasure or pain, affection, pity, fear, social proclivities, love of property, regard for rights and duty, obedience, shame, esthetic feelings, curiosity, and the like.

Pleasure and pain are indefinite or absent sensations in idiots, felt to a greater extent by imbeciles, and well marked in the feeble-minded. Joy, sadness, and anger are usually aroused by physical sensations. The self-mutilation of some idiots points to an absence of the pain sense, and idiot women have been known to bear children without experiencing the pains of labor. Idiots often cry out suddenly, burst out laughing, or throw themselves about, which is probably explicable by variations of perception in the somesthetic sense. Moral pain or remorse, usually wanting, is sometimes developed to a slight extent. It is not often that these defectives weep, and if they cry, it is but for some momentary pain or deprivation. They live in the present only, and do not concern themselves about the past or future. In the higher grades it is physical, seldom moral, pain that is taken note of. Pleasure is as little experienced as pain in the lower degrees, and laughter is as infrequent as crying. Pleasure is expressed by imbeciles and the feeble-minded by laughter, clapping the hands, or cries, though laughter, even with these, is uncommon. There are, however, certain imbeciles that always have a good-natured smile, and laugh readily and excessively over nothing. Frequently the laughter is a true automatic movement, as infantile spontaneous motor expression.

Affection is a sentiment not uncommon in idiocy, though it varies with the degree, being often rudimentary, vague, indefinite, and probably inspired rather by the ministration to his wants than by the caretaker. It is found that nearly all forms, except the lowest, appreciate kindness and patience, and are repulsed and made unmanageable by brusqueness or cruelty. With certain imbeciles and feeble-minded, where the moral sense is not too much obtunded, true affection for individuals is manifested; but when the moral sense is deficient, affection is elementary or absolutely wanting, so that kindness is either unappreciated or at once forgotten.

There are variations of the same nature in *love* for the family and in *friendship*. Absent in the simplest idiots, it may be shown in greater or less degree in the higher grades. In some it is unstable, changeable, and influenced much by the selfishness of the individual. In others, again, there is a perversion of family love, so that they are hateful and disagreeable to their parents or brethren. It is much the same with friendship. Often mild types of idiocy form in asylums friendships for one another, though they are too often apt to be associations of a sexual nature or for the purpose of combining together for mischievous purposes. A true solidarity of interests or social proclivity is seldom observed. Maltreatment of animals by idiots is usually due to ignorance, but there are moral imbeciles who perpetrate cruelties on animals as well as human beings from pure perversity and love of inflicting pain. The passion of *love*, when it exists, which is extremely rare, is founded altogether upon a physiological basis. *Jealousy* is sometimes, though infrequently, observed.

Pity is quite unknown in all degrees of idiocy. Some are amused or curious and some alarmed at the sufferings of others.

Fear is a common sentiment in all types of cases, more common than in normal persons, because of the want of understanding. Often the simplest occurrences inspire fear. On the other hand, when much excited, there are types that exhibit no fear at all.

Courage is wanting in all classes of idiocy. *Anger* is apt to manifest itself in all degrees and in every age. It is apt to be both causeless and paroxysmal, and to lead to the infliction of injuries upon the individual himself, upon inanimate things, or upon persons in the vicinity. The ungovernable rage is usually increased by efforts to restrain the patient.

Acquisitiveness is shown in imbeciles and the feeble-minded by a propensity for the collection of all sorts of useless objects and trifles, much the same as in cases of chronic mania. There is often a marked tendency to steal, sometimes deliberately, and at other times without motive, merely to gratify the desire of possession. The lower orders appropriate everything coming in their way, having no regard for the property of others. Many can be taught acquisition as a reward for labor, and, on the other hand, there are some who can be made to work only through fear, having, as they do, an innate antipathy to occupation of any kind.

With respect to *rights and duty*, the perceptions of the idiot vary with the degree of mental and moral defect. In some even inferior idiots these perceptions may be present, while with some the rights of others are never respected, though to their own they may cling tenaciously, and the feeling of duty may never be instilled into them, because of more or less moral perversion.

Obedience and *respect for authority* vary, too, with the amount of intelligence and the degree of moral impairment. Quite simple idiots may quickly respond to the word of command. On the other hand, some of the most intelligent may perversely resist all attempts at discipline. Compensation and punishment affect them variously. Reward in objective shape or in the form of praise is seldom appreciated by inferior grades, and often unduly by the higher. Punishment, objective or in the form of blame, is useless for the simpler degrees of idiocy, where acts are unintentional, and in some of the more intelligent excites antipathy, an unreasonable sense of injustice, and often causes them to harbor a vengeful feeling.

A true *religious sentiment* is quite unknown in any form of idiocy. This is true also of the feeling of *shame*. The only esthetic sentiment found in these defectives is the *love of music* or rhythm, which is quite general among all classes, though not perhaps so noteworthy as it has sometimes been stated to be. Occasionally we meet with cases having unusual musical aptitude. It is rather a rhythmic noise which appeals to most of them, such as beating of a drum, hammering, the grinding of an organ (even if out of tune and discordant). They have no sense of beauty, but things bizarre, grotesque, glittering, and colossal attract their attention. *Curiosity* and *astonishment* are aroused more readily through the sense of sight than that of hearing, and are often more easily roused in some of the lower grades than in the higher types of idiocy.

All classes evince a marked *credulity*, and often it is difficult or impossible to eradicate an idea once established. Fairy stories are especially pleasing to many of them, just as they are to children.

Veracity is a virtue which is uncommon among idiots. Many imbeciles are particularly apt to be untruthful and deceitful with regard to their faults, doings, physical condition, things found in their possession, and the like. Naturally, the simple idiot, owing to his feebleness of invention, if given to lying, limits his untruths to the simplest matters, such as denials of accusations brought against him, etc.

Physiognomy and Expression and Character.—Idiots all show deficiency in their general appearance. There is always something ungracious, uncouth, ugly in their figures, faces, attitudes, or movements. Very common among them are misshapen or asymmetrical heads, dwarfishness, lack of proportion of the limbs, stooping and slovenly postures, deformities of the hands or feet, and awkward and wobbling gait. The expression of the face varies from complete apathy and absence of intelligence to a considerable play of features of a low order, such as constant laughing, making faces, leering, or scowling. Besides the absence of those facial traits which are made on the face by the mind, the ugliness is generally added to by asymmetry, disproportion or deformity of the features. The eyes may be too close together or too far apart, or deformed by disease of the iris, cornea, or lids, or by squint. The nose deviates or is malformed, the ears are unshapely and unequal, the mouth half open, the teeth diseased and neglected; the chin deviated, prominent, or retreating; the forehead low and bulging or inclined. Microcephalus, hydrocephalus, and cretinism give their own ugly individuality too well known to need description here. Where a head is shapely and a face has any vestige of pleasing lines, it is generally fair to infer that the mental state is due to deprivation of one or more senses, or to the insanity of childhood.

As to *character*, this, too, varies with the amount of mental defect, and is difficult to analyze. In profound idiots there are often sudden accesses of excitement without apparent cause. In higher types the basis of character is inconstancy, weakness of will, and blunting of the sensibilities, their humor depending largely upon their environment, showing an appreciation of kindness and resentment of ill-usage. Some are clever and good-natured and funny, often making sharp remarks or doing amusing things, and at one time such cases were in great demand as court or family fools. History shows there were two kinds of fools made use of by royal and noble families—the true or natural fools (idiots or imbeciles), who were the first to create the profession, and their crafty imitators, the artificial fools, who made of it a profitable calling.

I should differ entirely from Sollier in his somewhat extraordinary distinction of imbeciles from idiots. He really selects one type of imbecile, while we know that there are many, and erects this single type into a great class which he everywhere distinguishes in his book as the imbecile. To him the imbecile is egotistical, boastful, vicious, careless, dangerous, a glutton, a vagabond, a mischief-maker, a sexual pervert, unstable, lazy, abusive, obscene, forgetful of kindness, vengeful, shameless, and altogether antisocial.

Language.—The primitive physical basis of language in the normal human infant is the auditory tract and the word-hearing center. It is essentially receptive. Then develops the word-comprehending center. After this the motor speech center is developed and associated with the primitive physical basis, thus establishing the emissive faculty. This rudimentary linguistic apparatus is variously defective in idiots. A defect in the emissive power is not so serious, as regards intelligence, as one in the receptive; for idiots of considerable intelligence may not be able to talk at all, while others very inferior may speak with readiness. Any part of this original physical basis of language may be affected, and the result to the defective individual will depend much upon what function is lost. The auditory apparatus may be imperfect.

The word-hearing center may not act. The word-comprehending c ter may be undeveloped. In such instances the intellect will su severely. Unlike the normal child, which comprehends many thi said to it as early as nine months of age, in cases of this kind comp hension will develop very late, or perhaps never ; yet occasionally w the development of the emissive power (without the word-comprehen ing center) words may be heard, learned, and repeated, constituting echolalia—speech without idea. Supposing the emissive apparatus al to suffer, we have hearing and comprehension and the development the mind, yet without the power of speech.

Like an animal, the idiot may be intelligent, but speechless. T development of language and intelligence is not parallel. Sollier c tinguishes two kinds of mutism in idiots—a motor and a sensory apha In the first he can not talk, though he understands ; in the seco nothing which is said is understood. Language is very late in devel ment in idiots. The crowing of the normal infant is not often observ but meaningless and monotonous cries take its place. The laryng sounds are earliest and best enunciated, the lingual and labial latest a least distinctly. Wildermuth classifies the dysarthrias and lalopath of idiots into two groups :

1. Where the disturbance of speech is the direct expression of t intellectual density. They lack ideas, and consequently have not t words for the expression of them. In the lowest degree, the idiot is vegetative automaton ; in a less profound degree, he is like a child two or three years, with imperfections of grammar and syntax.

2. When the disturbance of speech is a complication of idiocy, a is mechanical rather than intellectual, Wildermuth has rarely fou stumbling speech in the idiot, and never stammering. These defe are sometimes found in imbeciles, who, moreover, talk a great deal a without definite object ; who have onomatomania, and who are subj to transitory attacks of excessive and maniacal loquacity.

Considerable loquacity is occasionally observed in cases of acqui idiocy.

Next to hearing, the visual tract and the word-seeing and comp hending centers form a great receptive avenue for language and ide *Reading* will be impossible to such idiots as have defect of the vis apparatus or these centers, and the degree of acquisition of this pow will depend upon the degree of defect. There are idiots who lea merely the letters, others who acquire monosyllables, and still oth who can be taught to read laboriously. Sometimes such reading purely automatic, without actual comprehension. The higher the gra of idiocy, imbecility, or feeble-mindedness, the greater the develo ment of this faculty, though few of either class ever attain to perfec correct reading.

The *writing* center and its association tracts are the latest portions the linguistic cerebral basis to be established in normal cases, and the idiot are apt to be the least well-constituted. In addition to its i tellectual side, there is a complicated muscular coördination required writing which also renders it more difficult for defectives of this kin

They may be taught to reproduce letters, but the characters are meaningless to them. A few write quite legibly, though seldom or never well. As Sollier says, their writing is in reality drawing, and they like to copy printed letters, curved lines, and so on. There is a certain tendency to write with the left hand and to write from right to left.

In *drawing*, such as learn at all copy slowly and uncertainly, without perspective, and never draw without a copy or model ; or they do the work impatiently, and, if given free rein, indulge in curious and fantastic scrawls, such as are figured in the works of Sollier, Bourneville, and others.

Intelligence.—Since intelligence depends upon the acquisition, conservation, association, and production of ideas, and these upon the condition of the sensory organs and centers and language centers, it is mainly in intelligence that the idiot deviates from normal man. The deviation varies much in degree, from almost total absence to a condition nearly approaching the normal. The idiot has fewer ideas than the imbecile, and the imbecile fewer than the feeble-minded. All classes acquire ideas primarily in the same way as the normal infant—through the senses ; but while the normal child later on acquires ideas chiefly by means of language and imitation, the defective continues to make use mainly of the senses for this purpose, owing to the faulty development of the language centers. Preyer shows that questions and names are understood before the normal child can speak (nine months), while idiots, many years of age, may have an intelligent idea of the use of things, yet not know their names when heard, and be unable to speak them.

As regards concrete ideas, such as the different qualities of matter, it is noticeable that the idiot appreciates colors (particularly red), recognizes surfaces, avoids obstacles, and notices the difference between round and square, while distances and space are not comprehended. As Sollier correctly says, imitation, which is a source of ideas for infants, does not develop the intelligence of the idiot ; for to him it does not furnish an idea, but creates a mechanism. In the superior grades of idiocy imitation creates an idea which is assimilated by the intelligence ; but as the intelligence can not retain it, the result is the same as though it had not been assimilated. Still, it is not just to infer, from lack of intellectual expression, that there is complete intellectual inactivity. That ideas may exist in a brain apparently inactive is shown by the phenomenon of intellectual manifestation induced in idiots by severe pain, disease, etc. In other words, the intellectual receptivity of idiots may be greater than supposed, until some irritation occurs strong enough to show that the preceding stimuli have left their effects on the brain centers. Thus, Griesinger reports the case of an idiot who could only speak a few words until he contracted hydrophobia, when he began to talk of events which had taken place several years before.

As regards the *conservation of ideas*, we must remember, says Sollier, that memory is hereditary, organic, or acquired. Hereditary memory is extremely complex and difficult of explanation, but it apparently occurs in idiots. Organic memory, or unconscious memory,—viz., of

associated movements, such as walking,—although sometimes compl
sent in idiots, owing to defective nerve centers and lack of atte
, nevertheless, better developed than either of the two other vari
For acquired memory, attention is still more a *sine qua non*, and c
quently this is the least developed form of memory in idiots. Me
in an idiot develops slowly; at first its existence is shown only b
stimulus of some violent excitement. This indicates that memory
in so far as the conservation of the image is concerned, but not enoug
its reproduction under ordinary circumstances. In a higher degr
the development of memory, the idiot can recall the memory pictu
seeing again the original object (memory for food, memory for pl
Local memory, which does not act by satisfaction of a natural ne
only found in educable idiots (remembers his own bed, etc.).
memory is fixed by repetition of the sensation, and has not an emot
basis. These varieties of memory are simple, and do not neces
language. As soon as language exists, a much wider field opens fo
memory.

In simple idiots there is no association of ideas. The prim
forms of association, such as fear and the hope of reward, awake
associated ideas in them, and even in the superior types of idiocy
is no great development of this form of memory.

It is a curious and inexplicable phenomenon that in certain cas
idiocy there may exist particular, specialized memories, such as
musical airs, dates, and numbers, although memory, in its usual
general sense, may be deficient. Indeed, as a rule, the memory is f
in all classes of idiocy, and even in cases where the memory is f
well constituted it is ordinarily mechanical, useless to the posse
automatic.

Naturally, as abstract ideas result from reason, comparison,
judgment, such ideas are absent in the lowest order of idiocy.
found idiots have no idea of differences of persons or things. Hi
idiots may be able to appreciate superficial resemblances and differe
especially of color and form, but the discernment is so faulty
incorrect inferences frequently result.

Superior idiots appreciate resemblances more readily than differe
Simple generalizations may be possible, however, to all classes. In
lower types such generalizations occur only after long instruction,
once this power is acquired, they may be fairly correct, but in man
the higher they are hasty and often faulty. In educable idiots,
those who can not talk, there is an appreciation of number, and
may be taught to count. Addition is more easily learned than subt
tion, and multiplication can only be learned by those with f
developed memories. Division can rarely be taught them, and nei
idiots nor imbeciles can understand problems. The superior order
idiocy can count automatically, but rarely are able to do so with pr
understanding. They can say two and two make four, four and
make eight; but ask them how many are four and three and they
at sea. To count beyond ten, the number of the fingers, is ra
learned. But there are phenomenal instances where the mathema

faculty is remarkably developed, as in the cases of the so-called "calculating boys," some of whom, it is true, are normal in other respects, but many of whom are mentally defective, belonging to the category of idiots or imbeciles.

The idea of *time,* past and future, has seldom a place in the brain of the idiot.

Ideas in the idiot are too feeble to be fixed ideas, and while the higher types are sometimes subject to morbid impulses, there is not a true fixed idea, with consciousness and pain. With them such ideas should rather be called tenacious ideas.

The *association of ideas* occurs by resemblance, contrast, and contiguity. In the profound idiots, with few ideas, there may be an association of them in a very simple way—viz., the sight of food is associated with the sensation of satisfied hunger, and so awakens the idea of eating. It is an association of sensations rather than of ideas. The association of ideas should arouse the critical faculty. The *judgment* and *reason* in idiots are very faulty. They are founded on an association of few ideas, lack precision and firmness, and find their expressions in ambiguous language. A judgment is not always the result of reasoning. For reasoning, there must be some obstacle to an immediate conclusion. Justice, promptitude, and firmness, which are qualities of judgment depending on the attention, are lacking in the judgments of idiots. The idiots judge very falsely on account of lack of attention and of an association of the simplest ideas. All their sense illusions give rise to false judgments. Firmness is lacking in their judgments, as they have so little interest in what they decide upon.

Many imbeciles and feeble-minded, however, maintain their judgments with tenacity. They often have a very high opinion of their own intellectual faculties. This presumption leads them often to extreme blunders. If one of their judgments is admitted to be just, they become very proud of it, and immediately set to work to form others, which are generally absurd. Doubt which suspends action is rarely seen in any form of idiocy. The first impression capable of forming for them a judgment is followed immediately by the act, like a true reflex. Syllogistic reasoning does not occur either in idiots or imbeciles. Errors of the senses proceed from the perceptive apparatus rather than from the sensory apparatus. Since in idiots and imbeciles sense perceptions are retained in brain centers either undeveloped or diseased, and the memory pictures are consequently either confused or false, the association of these pictures is consequently faulty. In idiots, as the images are weak, the perceptive reasoning is also weak or wanting. In the imbecile, where the images are more numerous, the association may be falsified by a badly acting perceptive center. In him the association occurs so often by contiguity, and consequently the deduction is very liable to be erroneous, as contiguous ideas are not necessarily related; hence, incongruous observations and unexpected actions.

Sollier emphasizes the difference between idiots and imbeciles, which may be seen in the delirium sometimes occurring in these cases.

Exceptional in the idiot, when it occurs it is always in the im
form, unprovoked and without motive. It is a delirium of ac
imbeciles there are attacks of maniacal excitement, with impul
kill, to set on fire, or to break.

With respect to the *production of ideas*, there is little or none
inferior types of idiocy, and in the higher grades the imagin
inchoate, of no utility, and often directed to things that are evil.

Will, Personality, and Responsibility.—The elder Seguin
upon defect of will as the basis of idiocy, but the will is rather a
than a local function of the brain. It has no definite seat
encephalon, lesion of which would impair or destroy it. As
says, will in its simplest form is manifested by actions accomplis
the satisfaction of natural needs, appetites, and desires. Accor
the individual must have a consciousness of those needs. Such
sciousness may be very much blunted in profound idiots, and
quently the will will be almost entirely lacking. Such an idi
spinal being, and his movements may be compared to the refl
nomena seen in decapitated frogs. In higher idiots, the will i
fested by more complex movements, which are, however, cap
becoming secondarily automatic. Voluntary control of the sp
occurs only in idiots who learn to walk, and not until they have l
Volitions do not exist in the lowest order of idiots. The most
desires and the most primitive instincts are absent. The first to
is desire for food, but it may manifest itself simply by a str
out of the hand or a cry. In idiots in whom the will is more dev
and also in imbeciles, it finds its expression more easily in actio
in inhibitions.

Self-respect, very little developed in the idiot, plays a very im
rôle in the psychology of the imbecile, and by catering to it
often be made to do things which would otherwise be impos
obtain.

Intellectual movements, or acts accomplished under the influ
judgment or reason, are infrequent in the idiot, and not common
higher grades. Many idiots are incapable of choice. When the
of choice is present, it is often exercised with difficulty. He d
quickly understand that of two things he must take one an
the other—he wants to take them both. It is the same with
Between two desirable objects, the superior type does not hesit
takes without reflection the one he sees first, which he may
exchange when he sees the second.

In idiots, whose will and motor volitions are so feeble, *sug*
produces little or no results. It is the contrary in many im
except in those whose voluntary impulsiveness is too great. Ord
the higher grades are very susceptible to suggestion, as is seen
facility with which mischief is done by a band of imbeciles whi
been led on by one of their number. If suggestion is possi
imbeciles, it shows that the ideas which they already possess ar
unstable, and are easily replaced by new ones. It has a great a
with the suggestibility of the hysterical.

Consciousness and Personality.—As consciousness is but a phenomenon added to psychic processes, and not producing them, and as the personality is the coördination of psychic acts, it is necessary to form by deduction our conclusions as to these two attributes in the class of people we are studying. In absolute idiots it is not probable that any act is accompanied by consciousness. In higher idiots, in whom life is but little more than a succession of disconnected moments, it is not possible to say whether they have consciousness or not; but the personality, if present, must be very rudimentary, since an essential of its existence is a proper appreciation of the continuity of events.

For an individual to have consciousness of a psychic act, it is necessary that the exciting stimulus have a certain duration and intensity. Such factors in the stimuli are generally wanting in idiots; and so it is probable that most of their psychic phenomena occur without consciousness; and if there is consciousness, it must be very feeble. The distinction between the ego and the non-ego is not made by absolute idiots, and is but feebly present in the higher idiots.

In many imbeciles consciousness may be wanting or feeble, but in some it is clearly present, together with a perfect idea of their personality. Further, sometimes in delirium they have ideas of grandeur, showing an exaggerated conception of personality.

Responsibility.—All lower types of idiots are unable to manage their own affairs or to enjoy their civil or political rights, but those of a higher degree, who are at liberty, may have these rights.

Psychological Evolution.—In every degree of idiocy there comes a time, as Sollier well says, when the education stops and further mental progress ceases, and when the only hope is to retain the results which have been gained. This acme of development varies for the different psychic functions, so that one faculty may still improve, while another has already reached its cessation point. The senses continue to develop for the longest time, then the sentiments, and the intelligence the shortest. This is true of all classes, though the periods are longer in the higher grades, where all of the faculties are more equally and proportionally developed. Thus, in inferior types intellectual progress may cease at the age of six or seven, and the sentiments and senses continue their development to eighteen or twenty, while in superior grades the improvement of senses, sentiments, and intellect may cease about the same time—viz., at puberty.

Sometimes the faculties remain stationary, at others they retrograde when the limit of development is reached. Retrogression follows the same law as dementia—namely, progressive enfeeblement of will, intelligence, sentiments, and sensations, in the order named. When retrogression begins in the simpler forms it is very rapid, but in the higher types goes more slowly and more irregularly. Purely intellectual gifts which they have acquired (reading and writing) disappear very rapidly. In the intellectual downfall of the superior types one sees from time to time flashes of intelligence, like reflections from their weakening minds, but such are not observed in the lower forms.

General Pathological Anatomy.—There has been accumulated in literature of late years a great deal of valuable matter relating to the pathology and morbid anatomy of idiocy, so that much new light has been shed upon a somewhat obscure subject. The investigations of Sachs and myself[1] into the causation of the cerebral paralyses of children, which are so frequently associated with the various degrees of mental impairment, from feeble-mindedness to profound idiocy, and in which we found meningeal hemorrhage to be so commonly the primary lesion, might well give rise to the belief that in a majority of cases of idiocy without paralysis and in idiocy associated with epilepsy we are confronted with the same initial lesion. The site of the meningeal hemorrhage is the determining factor in the establishment of the symptoms. If the Rolandic area be mainly implicated, either on one or on both sides, we have a hemiplegia or diplegia as the result, and these paralyses may be severe or light according to extent of the hemorrhage, and may be associated with idiocy or epilepsy, depending also upon the extent of the lesion and upon the amount of irritation. Again, I have seen a case in which there was left hemianopia, epilepsy, and very slight mental impairment, pointing to a meningeal hemorrhage over the right occipital lobe. Probably, too, some of the cases of arrested development of the speech, with or without enfeebled mind, are due to the same cause. It may be assumed also that meningeal hemorrhage often occurs as the initial lesion in what appears to be idiopathic epilepsy. The symptom or syndrome produced then will depend upon the location and extent of the initial lesion. Asphyxia at birth and convulsions shortly after birth are in themselves significant of meningeal hemorrhage, and in our study of etiology we observe the great frequency of these symptoms in the history of idiocy. At our autopsies, which are nearly always made years after the initial lesion, we find only terminal pathological states, such as atrophy, general sclerosis, and cysts, and, unfortunately, these conditions are not pathognomonic of antecedent hemorrhage, for they also are the terminal states for embolism, thrombosis, cerebral hemorrhage, meningitis, and meningo-encephalitis. What other evidence have we that proves the enormous preponderance of meningeal hemorrhage in the etiology of the terminal pathological conditions? It is in the testimony of the investigators of the causes of still-birth. For instance, Litzmann[2] examined 161 stillborn children, finding in them 35 cases of meningeal hemorrhage. Parrot,[3] in 34 autopsies on the new-born, found 5 with blood in the arachnoid cavity and 26 with hemorrhage into the subarachnoid space.

The study of Sarah J. McNutt,[4] of New York, in 1885, of 10 similar cases added valuable testimony to that already given, and showed the relation between meningeal hemorrhage and asphyxia and convulsions in the new-born in a manner not to be gainsaid.

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[1] "The Cerebral Palsies of Early Life, Based on a Study of One Hundred and Forty Cases," "Jour. Nerv. and Ment. Dis.," May, 1890. See also paper on same subject by author, Louis Starr's "Text-book of Diseases of Children," Phila., 1894, and Sachs' "Nervous Diseases of Children," New York, 1895.

[2] "Archiv für Gyn.," Bd. xvi, 1880.

[3] "Clinique des Nouveau-nés," Paris, 1877.

[4] "Amer. Jour. of Obstetrics."

Allusion is elsewhere made to Herbert R. Spencer's 130 autopsies in still-born children, in which there were 53 instances of hemorrhage from the pia and arachnoid.

Thus, the evidence before us in favor of meningeal hemorrhage as the initial lesion in a large proportion of cases of idiocy is most convincing. Some idea of the character of the terminal states found in idiocy may be derived from the studies of Wilmarth[1] and Bourneville.[2] The former communicates the results of 100 autopsies, which he summarizes as follows:

Sclerosis with atrophy, 12; sclérose tubereuse, 6; diffuse sclerotic change, 7; degenerative changes in vessels, ganglionic cells, or medullary substance, not constituting true sclerosis, 15; hydrocephalic, 5; general cerebral atrophy, 2; non-development in various forms, 16; infantile hemorrhage, 1; extensive adhesion of membranes from old meningitis, 3; angiomatous condition of cerebral vessels (with degenerative changes), 1; glioma (with sclerosis), 1; porencephalia (with non-development), 1; of 31 cases where actual disease or imperfect development of the brain proper was not demonstrated, there was hypertrophy of the skull, 6; acute softening (recent), 2; demimicrocephalic, 2; when the brain was above usual weight, but the convolutions large and very simple in their arrangement, 2.

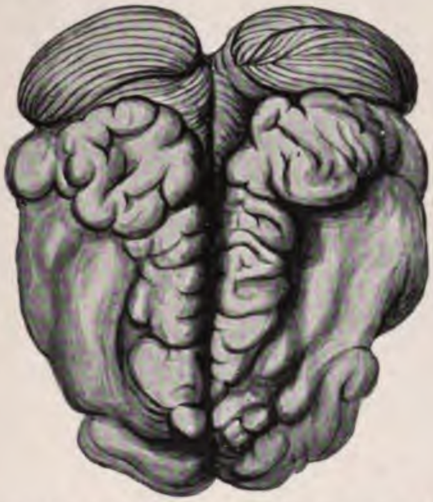

Fig. 336.—Brain of a diplegic idiot, showing atrophy of the convolutions over large symmetrical areas —not a true parencephalia. (See history of case, "Proc. N. Y. Path. Soc.," 1894, p. 94.)

Our examination of this summary discloses the fact that atrophies and diffuse sclerosis were demonstrated in 21 of the cases and tuberous sclerosis in 6. It is probable that the tuberous form of sclerosis has a pathology different from that of the diffuse form and more resembling the disseminated sclerosis of neuropathologists. Fifteen of Wilmarth's cases are recorded as presenting degenerative changes in vessels, ganglionic cells, or medullary substance, "not constituting true sclerosis." There was evidently some resemblance to sclerosis, or this author would not have qualified his description thus; and it is more than probable that the condition would have been pronounced one of genuine diffuse sclerosis by experts at the present day. Wilmarth notes 16 cases of non-development in various forms. He writes, in this connection:

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[1] "Proceedings Ass'n Amer. Inst. Idiots and Feeble-minded," 1891.

[2] "Recherches sur l'epilepsie, l'idiotie," etc., Paris, 1880–1897.

"Non-development is found in several forms. A portion of the cortical substance may be thin, and, instead of following the typical arrangement of the fully developed brain, form a number of irregular folds, which may be so small and numerous as to resemble a mass of angle-worms."

This is evidently the condition which we know as microgyria, a true pathological process probably due to a vascular lesion (thrombosis or embolism), and not, therefore, a fault of development. Wilmarth's observations were made, many of them, years ago, before neuropathology had attained its present precision, and hence have not the value of later researches, such as those undertaken at Bicêtre and Upsala.

Hammarberg[1] has made one of the most valuable contributions to the study of the pathology of idiocy in literature. His study enters into the details of the examination of the brains of nine cases of idiocy, imbecility, and feeble-mindedness. Several of these were epileptic and paralytic idiots. His pathological investigations were controlled by the microscopic examination of twelve normal brains. The results were briefly as follows: In all of the cases of idiocy a more or less large part of the cortex showed arrest of development at a stage corresponding to either an embryonal period or the period of early infancy. Only a small number of cells reached their higher development or were destroyed during the growth of the cortex. The mental defects were in direct proportion to the defects of the development of the cells, and were greater the earlier the period of arrest of development.

As regards hydrocephalic idiocy, the true pathogeny of hydrocephalus is unknown. It is generally explained as being due to a chronic intraventricular meningitis, a congestion of the ependyma. But in many of these cases nothing abnormal is observed about the ependyma save thickening. It is possible that a careful study of the manner of secretion of the cerebrospinal fluid and of the relations existing between the ependyma and the external serous membrane of the brain may help to elucidate the origin of the disorder; for there is some reason for believing that a sort of current of fluid flows from the ventricles into the exterior serous cavity through the foramen of Magendie, the foramina of Mierzejewsky, and two other foramina which have been described, but are of uncertain existence. The ventricular walls secrete the cerebrospinal fluid and the exterior serous cavity absorbs it, according to this theory. Thus, then, there may be three processes by which primary hydrocephalus may be induced: hypersecretion in the ventricular spaces, occlusion of the foramina mentioned, and disorder of the absorbent apparatus. An interesting study of the subject along this line might be made.

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[1] "Studien über Klinik und Pathologie der Idiotie," by C. Hammarberg, Upsala, 1895.

When the fluid begins to increase in the ventricles, these become dilated, as a rule equally, occasionally unequally, from obliteration of the foramen of Monro. The dilatation may be restricted to the lateral ventricles, or may include the third and fourth also. With the distention of the ventricles compression of the brain-substance takes place, giving rise to functional impairment of various kinds and degrees. With increase of pressure, atrophy of the compressed parts occurs. The septum between the ventricles may disappear and the brain-envelope become thin as paper, so that the hydrocephalus is like one enormous cyst filling the cranial cavity. The basal ganglia and brain-stem become flattened. Examination of the cerebral envelope shows atrophy and degeneration of cells and fibers. The distention may go on until the cerebral tissues and the membranes vanish almost entirely. The amount of fluid has been known to reach six, eight, ten, twenty, and even twenty-seven pints. The following is an instance in point (a case from the Randall's Island Hospital for Idiots, the autopsy of which I reported at the New York Pathological Society. See "Proceedings," 1894, p. 94):

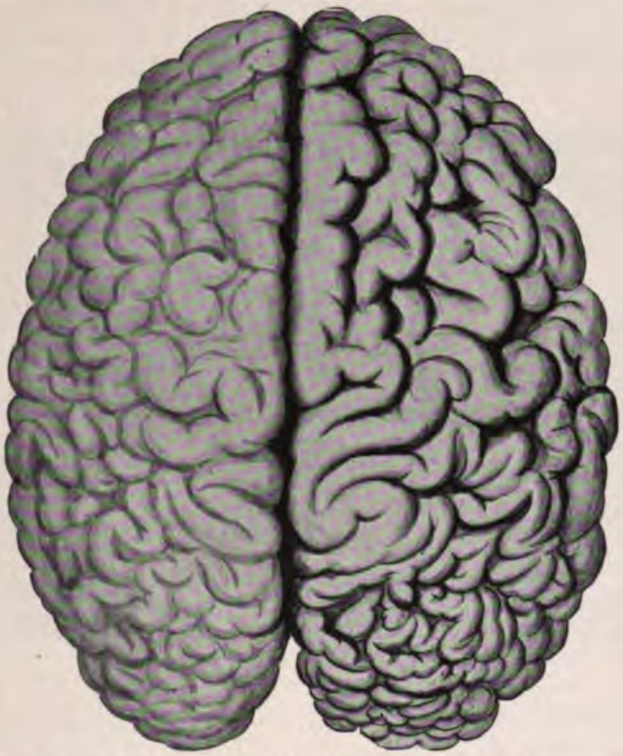

Fig. 337.—Brain of a blind hemiplegic idiot. Atrophy and microgyria in both occipital lobes. (See history of case, "Proc. N. Y. Path. Soc.," 1894, p. 98.)

A female child, aged eighteen months; hydrocephalus, whether congenital or acquired unascertained. Circumference of head, 51.5 cm.; anteroposterior diameter, 18 cm.; greatest transverse diameter, 15 cm.; naso-occipital arc, 32 cm.; binauricular arc, 34 cm.

Blindness and nystagmus; widely gaping fontanels; spastic diplegia; occasional convulsions, and just before death opisthotonos. At the autopsy sixty-four ounces of reddish serum were first removed by tapping the anterior fontanel. The skull and dura were exceedingly thin. The falx cerebri had disappeared. Cutting through the thin dura, nothing was to be seen of any brain proper in the great cavity of the head. The membranes usually covering the cerebrum had disappeared with that organ. At the base of the skull the floors of the ventricles and basal ganglia stood out prominently, and back of these parts, lying on the tentorium, were the only vestiges of a cerebrum—parts of the two occipital lobes. On removing the tentorium, the cerebellum was found to be of about normal size. Microscopical examination showed degeneration and atrophy of the lateral columns of the cord (Fig. 338).

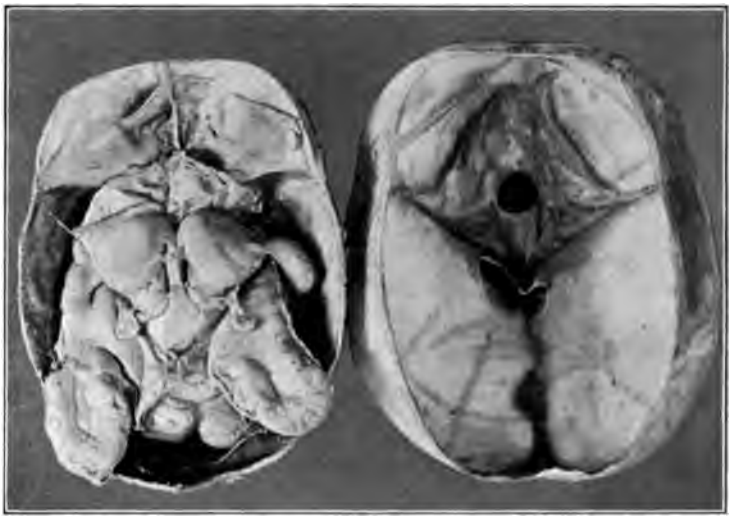

Fig. 338.—Brain and skull in a case of hydrocephalus.

In this case, then, we have to do with distention and atrophy of the encephalon pushed to its greatest extreme.

Case IV, in a series of autopsies by Bourneville, is a good illustration of the nature of the process of compression and atrophy. A girl, a complete idiot, died at the age of about two years. Five hundred grams of fluid were found in the brain-cavity, the brain-envelope having become merely a sac of varying thickness. For instance, in the right hemisphere, over the whole of the temporo-occipital region, the wall of cerebral substance was but a millimeter in thickness, and at one place here, near the fissure of Sylvius, the brain-substance was absent altogether at a space of four centimeters in diameter, closed merely by a fine meningeal veil. In this case, then, the process of complete atrophy of the brain was arrested by death.

As the ventricular cavities dilate, pushing the brain-envelope with them, the skull-cavity is distended and the cranial bones are separated, made thinner, and expanded in area. The enlargement of the head is directly proportional to the youth of the patient. Cases beginning before or shortly after birth will present greater expansion of the cranial cavity than such as have a later origin. Sometimes some sutures give way and others become synostosed. Where sutures are separated Wormian bones often form, or a membranous connection is established between the cranial bones.

Occasionally, in these cases of primary hydrocephalus, the defects of brain-substance are not due to pressure-atrophy, but there is an associated condition of malformation or defect. Thus, in an autopsy of Bourneville's, on a girl about thirteen years of age, with congenital hydrocephalus, idiocy, and epilepsy, the hemispheres of the cerebellum were totally absent, the cerebellum being represented by the vermis, which was the size of a pigeon's egg. Perhaps such a defect is due to a pressure-atrophy beginning very early in fetal life.

As regards the pathology of secondary hydrocephalus, we possess more definite knowledge. In this the internal hydrocephalus is caused by obstruction of the veins of Galen, or by obliteration of the foramina of Monro, Magendie, or Mierzejewski. Common causes are tumors of the cerebellum, such as sarcomata and tubercles. Meningitis may act in the same way. The amount of hydrocephalus, ventricular dilatation, and expansion of the skull thus induced will depend directly upon the youth of the infant or child. As a rule, secondary hydrocephalus never reaches the extent of the primary form, owing to the rapidly fatal nature of its cause. In these cases we seldom see pressure effects beyond flattening of the convolutions and moderate expansion of the cranial vault.

An exceptional and an extremely interesting case was one upon whom I made an autopsy at Randall's Island, not long ago. It was a case of very marked hydrocephalus in a child of four years, in which a small tumor of the pineal gland, the size of a small hazel-nut, compressed and obliterated the aqueduct of Sylvius. Both of the lateral ventricles were enormously distended, the left more than the right, and contained twenty-four ounces of clear fluid. The third ventricle was also widely dilated. The fourth ventricle was of normal size. Microscopical sections of the quadrigeminal region revealed the obliteration of the aqueduct. The tumor was apparently tubercular, but was not examined, it having been mislaid and lost.

The cases of acute hydrocephalus due to meningitis serosa, and the cases in which a defect of brain-substance is counterbalanced by an equal bulk of cerebrospinal fluid, do not commonly fall under this heading.

In chronic hydrocephalus internus there seems to be a special susceptibility of the membranes to acute disease, so that at autopsy it is not uncommon to find evidence of an acute meningitis, simple, hemorrhagic, suppurative, or tubercular.

The fluid found in hydrocephalic idiots has been frequently analyzed. In a case of Bourneville's the analysis of the hydrocephalic fluid, withdrawn nine hours after death, resulted as follows: Color, pale yellow; aspect, clear after standing; reaction, neutral; odor, like that of blood; consistence, slightly viscous; density, 1.006; organic matter, 1.65; salts, 10; total fixed solids, 11.65; phosphoric acid, 0.22; sodium chlorid, 0.80; albumin, 0.26; leukocytes, very few; red blood-corpuscles, considerable.

In microcephalic idiocy we recognize three distinct classes:

1. Morphological microcephaly, in which there are no pathological changes in the brain, but simply a brain arrested in its development with persistent fetal morphology.
2. Pathological microcephaly, in which the small size of the head is determined by morbid processes in the brain (such as meningeal hemorrhage, thrombosis, porencephalic defects, etc.).
3. Mixed cases of microcephaly, in which pathological processes are superadded to or associated with true morphological microcephaly.

The following table gives a summary of the pathological conditions responsible for most cases of idiocy:

ETIOLOGICAL FACTORS.	PRIMARY LESIONS.	TERMINAL CONDITIONS FOUND AT AUTOPSY.
Hereditary degeneracy.	Developmental defects of portions of the brain, such as corpus callosum, one hemisphere or part of a hemisphere.	Same, with compensatory hydrocephalus internus, externus, or both; compensatory thickening of skull.
Hereditary degeneracy.	Micrencephalus, with or without defects.	Same. Brain-substance often sclerotic; deficient in microscopical elements. Sometimes compensatory hydrocephalus.
Hereditary degeneracy.	Agenesis corticalis; slight changes in gross appearance of brain; maldevelopment of microscopical elements.	Same. Sometimes hydrocephalus externus.
Vascular disorders of fetal brain.	Partial defects like porencephalia, microgyria.	Same. Compensatory hydrocephalus and thickening of the skull; atrophy and sclerosis of affected convolutions or lobes.
Diseases of mother or trauma to mother. Fetal disorders, such as syphilis, asphyxia at birth, prolonged labor, infantile convulsions, febrile diseases of child, cerebral diseases of child.	Meningeal hemorrhage; thrombosis; embolism; cerebral hemorrhage; meningitis; meningo-encephalitis.	Atrophy; diffuse sclerosis; cysts; meningo-encephalitis.
Uncertain fetal and post-natal causes.	Tumor sometimes; oftener unknown.	Hydrocephalus.
Antecedent infectious diseases of mother or child (?).	Tuberous sclerosis.	Tuberous sclerosis.

In amaurotic idiocy but six autopsies have been made, and thus far the changes found may be considered to be simply degeneration of the gray matter of the cortex and of the anterior horns of the cord (Sachs).[1]

Diagnosis and Prognosis of Idiocy.—Diagnosis of Idiocy in General.—It is seldom difficult to make a diagnosis of idiocy in childhood when the individual has reached such a stage of development that backwardness and deficiency stand out in prominent contrast to the normal average of intelligence in children of the same age. Occasionally, however, we have to deal with some species of insanity in childhood, in which case the matter of diagnosis is important because of the more favorable outlook for insanity. There are not a few patients cared for in institutions for the feeble-minded and idiots in which insanity has been the original factor in the mental impairment, and when the histories of such are obscure, it is almost impossible to distinguish between ordinary idiocy and what may be truly termed a terminal dementia following upon some acute insanity of childhood. In these cases residual symptoms of a psychosis can be our only guide.

The diagnosis of some form of idiocy in infancy is far from easy unless one familiarizes himself thoroughly with the manifold steps of development for the first few years of existence. Early diagnosis is of the utmost importance, not only for the benefit of the unfortunate child itself, but on account of the deep solicitude of the parents for its future. One of the chief aids in differentiation will be found in a study of the physical condition of the infant. The shape and size of the head should be carefully noted and compared with normal shapes and statistics. Unfortunately, there are no elaborate tables of head measurements in infants and children as yet made which can be looked upon as a final establishment of the normal averages, but the following figures are fairly representative of cranial measurements:

Circumference at birth	36 cm. in both sexes.
Binauricular arc	22 " " " "
Naso-occipital arc	22 " " " "

At the age of one year these dimensions have increased to—

Circumference	44 cm. in both sexes.
Binauricular arc	27 " " " "
Naso-occipital arc	30 " " " "

Malformation and asymmetry of the head should be taken into consideration. The various malformations are treated of in another chapter. The presence of marked anatomical stigmata of degeneration is of significance. Paralysis of a limb or limbs, if of cerebral origin, is of great importance, indicating, as it does, some lesion of the brain, which may retard or restrict mental development and lead to paralytic or epileptic idiocy, or both. Some of the morbid movements, such as nystagmus,

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[1] "A Case of Amaurotic Family Idiocy with Autopsy," by Frederick Peterson, M. D., "Jour. Nerv. and Ment. Dis.," July, 1898.

ataxia, chorea, or athetosis, may be present, and, as symptoms of
der of the central nervous system, should lead to a careful inve
tion of the whole mental and physical organization.

While it is frequent to find evidence of idiocy immediately a
rth in bodily and especially in cranial and facial characteristics,
ter careful examination as to imperfect action of the sensations
perceptions, we may sometimes recognize idiocy in cases where physi
evidence is wanting. The child may not learn easily to take the bre
Its cry is different from that of other children. It cries with
motive. Sometimes there is congenital blindness or congenital de
ness (there is always deafness in every child for several days a
birth). In the normal child the sense of smell may be stimulated i
mediately after birth, and taste is evident in a few days. In the i
these special senses may be retarded in their development, or abse
The movements of the eyes are generally irregular, and strabismu
frequent until the end of the second month in normal children, so t
in the diagnosis of idiocy this can not be relied upon as significant un
the eye-movements are imperfect after the third month. In the nor
child the eyes follow a light between the third and fourth weeks ; in idi
this ability may be retarded indefinitely. The normal child starts
gentle touches on the third day after birth. The new-born idiot may
immobile or feeble in its reactions to cutaneous stimuli. The nor
child laughs at tickling in the eighth week, while the idiot or imbe
is not incited to laughter ordinarily at all in the earliest years of li
From these facts it follows that in defectives we must examine the se
sory organs themselves, so far as possible, for defects, as well as stu
their reactions and impaired perceptions of sensations.

Preyer, in his work on " The Mind of the Child," gives a conspec
of the development of the normal faculties during the first forty mon
of the child's life, and the following brief abstract is made therefrom
purposes of comparison with the mental development of the idiot:

NORMAL CHILD.

First Month.—Sensitive to *light* as early as first and second da
Pleasure in light of candle and in bright objects on eleventh d
Hears on fourth day. Discriminates sounds last two weeks of mon
Starts at gentle *touches* second and third days. Sensibility to *taste* ab
end of first week. Strong-*smelling* substances produce mimetic mo
ments at birth.

Pleasure first days in nursing, in bath, in sight of objects.

Discomfort first days from cold, wet, hunger, tight clothing.

Smiles on twenty-sixth day.

Tears on twenty-third day.

Vowel-sounds in first month.

Memory first active as to taste and smell; then as to touch, sig
hearing.

Incoördinate *movements of the eyes*.

Sleeps two hours at a time, and sixteen hours in twenty-four.

Reflexes active.

Second Month.—Strabismus occasional until end of month. Recognizes human voices; turns head toward sounds. Pleased with music and with human face. Sleeps three, sometimes five or six, hours. Laughs from tickling at eighth week. Clasps with its four fingers at eighth week. First consonants from forty-third to fifty-first days (*am-ma*, *ta-hu*, *gö*, *ara*).

Third Month.—Sixty-first day, cry of joy at sight of mother and father; eyelids not completely raised when child looks up. Accommodates at ninth week. Notes sound of watch at ninth week; listens with attention.

Fourth Month.—Eye-movements perfect. Objects seized are moved toward the eyes. Grasps at objects too distant. Joy at seeing self in mirror. Contraposition of thumb in grasping at fourteenth week. Head held up permanently. Sits up with back supported at fourteenth week. Beginning to imitate.

Fifth Month.—Discriminates strangers. Looks inquiringly. Pleasure in crumpling and tearing newspapers, pulling hair, ringing a bell. Sleeps ten to eleven hours without food. Desire shown by stretching out arms. Seizes and carries objects to mouth. Consonants *l* and *k*.

Sixth Month.—Raises self to sitting posture. Laughs, and raises and drops arms when pleasure is great. "Crows" with pleasure. Compares image of father in mirror with original.

Seventh Month.—Astonishment shown by open mouth and eyes. Recognizes nurse after four weeks' absence. Sighs. Imitates movements of head, of pursing lips. Averts head as sign of refusal. Places himself upright on lap.

Eighth Month.—Astonishment at new sounds and sights; at imitations of cries of animals.

Ninth Month.—Stands on feet without support. More interest shown in things in general. Strikes hands together with joy. Shuts eyes and turns head away when something disagreeable is to be endured. Fear of dog. Turns over when laid face downward. Turns head to light when asked where it is. Questions understood before child can speak. Voice more modulated.

Tenth Month.—Sits up without support in bath and carriage. First attempts at walking at forty-first week. Beckoning imitated. Missed parents in absence, also a single ninepin of a set. Can not repeat a syllable heard. Monologue and hints at imitation (*mä*, *pappa*, *tatta*, *appapa*, *baba*, *tätä*, *pa*, *rrrr rrra*).

Eleventh Month.—Screaming quieted by "sh." Sitting becomes habit for life. Stands without support. Stamps. Syllable correctly repeated. Whispering begins. Consonants *b*, *p*, *t*, *d*, *m*, *n*, *r*, *l*, *g*, *k*, vowel *a* most used, *u* and *o* rare, *i* very rare.

Twelfth Month.—Pushes chair. Can not raise self or walk without help. Obeys command, "Give the hand."

Thirteenth Month.—Creeps. Shakes head in denial. Says *papa* and *mamma*. Understands some words spoken.

Fourteenth Month.—Can not walk without support. Raises himself by chair. Imitates coughing and swinging of arms.

Fifteenth Month.—Walks without support. Laughs, smiles, gives a kiss on request. Repeats syllables. Understands ten words.

Sixteenth Month.—Runs alone. Falls rarely.

Seventeenth, Eighteenth, and Nineteenth Months.—Sleeps ten hours at a time. Associates words with objects and movements. Blows horn, strikes with hand or foot, gives leaves to stag, waters flowers, puts stick of wood in stove, washes hands, combs and brushes hair, and other imitative movements.

Twentieth to Twenty-fourth Month.—Marks with pencil on paper, whispers in reading newspaper. Very few expressions of his are recognizable. Executes orders with surprising accuracy. Tries to sing and beat time, and dance to music.

Twenty-fifth to Thirtieth Month.—Distinguishes colors correctly. Sentences of several words. Begins to climb and jump and to ask questions.

Thirtieth to Fortieth Month.—Goes upstairs without help. Sentences correctly applied. Clauses formed. Words distinctly spoken, but influence of dialect appears. Questioning repeated to weariness. Approximates manner of speech to that of family more and more.

By contrasting the mental development of the supposedly abnormal child with these observations of Preyer upon normal development, it will not be difficult to appreciate impairment of varying degree. The presence of mere backwardness may not infrequently, however, be observed in children that later develop normally, and it is well to bear this fact in mind; but the combination of backwardness in the development of the sensations, perceptions, ideation, and speech with marked physical signs of degeneracy or brain lesion would be naturally of the greatest importance from the diagnostic point of view.

Diagnosis of the Form and Nature of the Idiocy.—While the diagnosis of the presence of idiocy is, as a rule, fairly easy, especially after infancy has reached the stage of childhood, the diagnosis of the type or kind of idiocy presented is often attended with great difficulty. Where the cerebral disorder or defect is accompanied by striking physical peculiarities or malformations, such as hydrocephalus, microcephaly, paralysis, or myxedema, we are immediately in a position to classify the type. In idiocy associated with epilepsy, too, we can readily approximate the type, though it must always be remembered that there are three distinctive ways in which epilepsy and idiocy are correlated—viz., paralytic idiocy combined with epilepsy, epileptic idiocy from a homologous lesion not implicating the motor centers or tracts, and, finally, dementia in childhood depending upon the epilepsy. The traumatic class of cases is recognized either by the external evidence of injury to the skull or by the history of direct relation of the psychic symptoms to the antecedent trauma. The sensorial type of idiocy is distinguished by existing or foregone loss of two or more senses, particularly blindness and deafness. The amaurotic type presents a characteristic syndrome—viz., flaccid or spastic weakness or paralysis of the whole musculature, diminished or exaggerated tendon-reflexes, distinctive changes in the fundus leading to optic atrophy, and marasmus. In the majority of cases, then, we are in a position to determine readily the form of idiocy presented by the patient and to formulate an opinion as to the nature of the pathological process or the condition underlying it; but there will still remain a considerable number of cases in which diagnosis can not be made during life, either as to the type of idiocy before us or as to the character of the process. Among such puzzling cases will be those indistinguishable from the psychoses of early life; idiocy following meningeal hemorrhage and meningitis without inducing either paralysis or epilepsy; idiocy due to tuberous sclerosis, and the like.

Diagnosis of the Degree of Idiocy.—It is necessary, for purposes of medicopedagogical treatment, to comprehend the degree of idiocy, not only to determine whether it is simple idiocy, imbecility, or feeble-mindedness, but to ascertain, as far as possible, the different shades of each of these; and it is useful, too, to watch the progress of a case under treatment, and to record from time to time the advance made by the patient and pupil. Accordingly, the writer has drawn up what may be termed a species of mind chart, as given opposite. The physician will be familiar with the ordinary tests for common and special sensibilities. The intensity and duration of attention may be studied, in the same connection, by methods which will readily suggest themselves in relation to objects, colors, sounds, smells, and tastes, which are utilized in such a way as to demonstrate perception, the retention of the perception, and the duration of such retention. The chief difficulty will be in determining and recording the purely intellectual features of the case; but some patience and perseverance will demonstrate the ability and degree of ability of the patient to acquire, conserve, associate, and produce ideas, concrete and abstract; to appreciate resemblances and differences; to count, add, subtract, and divide.

Prognosis.—As regards the cure of idiocy, there can not be any difference of opinion. There are few cases—indeed, almost no case—in which improvement to some degree may not be promised under proper conditions; but cure there is none. The profound idiot may be regenerated to some slight degree; be made less repulsive, less offensive, less destructive. The imbecile can be taught cleanliness, speech, divers occupations. The feeble-minded subject is susceptible of enormous improvement. It is impossible in any case to predict how much advance may be made under the best supervision, but it will be safe to say that the methods now in vogue in the training of the idiot will surprise the relatives or guardians by their efficacy, and there is no case so unpromising and hopeless as to contraindicate an attempt at improvement. Left to itself, even a mild type of idiocy will not only make no progress, but will be certain to degenerate, to lapse into a lower grade. Shuttleworth,[1] in reviewing the results of twenty years' experience at one of the large English institutions, states that of patients discharged

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[1] Tuke's "Dictionary of Psych. Med.," p. 675.

MIND CHART.

Name .... Age .... Sex

Constitution (feeble, fair, robust, or obese) ....

Form of idiocy .... Degree of idiocy ....

Paralysis, deformity, or morbid movements ....

Right- or left-handed .... Temperament (cheerful, gloom

sluggish, etc.) ....

Sense defects.	Sight.	Hearing.	Taste.	Smell.	Tactile and pain.	Muscular.
Intensity and duration of attention.						
Instincts.	Hunger.	Self-preservation.	Sleep.	Voluntary movements; play.	Sexual.	Imitation.
Morals and Habits.	Tidiness.	Destructiveness.	Humanity.	Veracity.	Politeness.	Obedience.
Sentiments.	Pleasure and pain.	Affection.	Fear.	Anger.	Acquisitiveness.	Shame.
Language.	Speech.	Reading.	Writing.	Gesture.	Drawing.	
Intellect.	Ideas.	Memory.	Association of ideas.	Reason.	Judgment.	Will.

Special aptitudes ....

therefrom after full training, 10 per cent. became self-supporting, another 10 per cent. might have become so had they obtained suitable situations, and about 20 per cent. were reported as useful to their friends at home. This bears out the earlier estimate of Seguin, who said that "more than 40 per cent. have become capable of the ordinary transactions of life under friendly control, of understanding moral and social abstractions, of working like two-thirds of a man; and 25 to 30 per cent. come nearer and nearer the standard of manhood, until some of them will defy the scrutiny of good judges, when compared with ordinary young men and women."

There are certain features in connection with the different types of idiocy which are helpful in forming our opinion as to the probable future of a patient. For instance, it may be taken as an axiom that the greater the defect or injury of the brain, the profounder will be the mental impairment and the more difficult will be the labor of bringing about an amelioration of the condition. The earlier, too, that the brain is hampered in its development, the worse, as a rule, is the prognosis. This holds good for every form of idiocy. Hence the outlook for the congenital types is less promising than that for the acquired, and for idiocy acquired in the first year less than that for idiocy acquired in the second. Some of the prognostic indications of the special forms will be discussed under their respective captions; but, in general, it may be assumed that microcephalic idiocy and congenital hydrocephalic and paralytic idiocy will be benefited least among the types of idiocy discussed, and always in proportion to the intensity of the morbid process. The sensorial, traumatic, and myxedematous forms are, *ceteris paribus*, among the most promising. The amaurotic form is generally fatal. Idiots with special aptitudes, or *idiots savants*, tend to early psychic degeneration. Idiots that are extremely restless, as shown by incessant motion of the hands, arms, head, trunk, or by constant walking, are generally among the most intractable, because of the difficulty of fixing their attention.

Although there is scarcely ever to be encountered an idiot in whom improvement of some kind can not be brought about by assiduous cultivation of whatever residual faculties and functions he possesses, it is practically necessary to classify idiots into teachable and unteachable. It is practically so because a majority of these defectives are found among the poor, who can not command all that the world affords in the way of treatment, care, and training. Nor could the commonwealth assume the enormous task of doing the best for all its idiot charges. No community could possibly be repaid for any such undertaking, because the idiots classified by public authorities as unteachable are not susceptible of such development as would satisfy the tax-payer's right to ask the utility of the expenditure. It is only with private families that anxious parental solicitude will and can command that medico-pedagogical care, skill and patience which can overcome almost insuperable difficulties in the education of profound idiots. Practically, therefore, we find that there is a tendency to separate idiots into the teachable and unteachable; a tendency in our public institutions to exclude the them to attain the age of forty and more years; hyd still earlier. The same is true of profound cases of myxedematous idiocy. The rare form known as a almost invariably fatal in infancy.

General Treatment of Idiocy.—The treatment volves the employment of both physician and teache medicopedagogic is made use of to designate this com cal and educational features for the care of the defec the union of the two professions for such purpose the relatively the higher and more important position. services of trained care-takers or nurses are not to be o patient will profit most who receives the properly com best physician, best teacher, and best nurse. As a ru concurrence of necessary aids is more apt to be found private institution than in the home; but that it is po treatment at home under favorable circumstances, is n

The methods of procedure formulated by Itard, exp and employed now-a-days everywhere in private and for idiots, with modifications induced by experience of educational science, are well described in the writin Shuttleworth, Ireland, Down, and others. A brief below of the process of

Education of Idiots.—The educational treatment soon as the diagnosis of defective intelligence is made. pushed vigorously at too early an age; but infancy, w system is most impressionable, plastic, and pliable, is t modification and the bringing out of the rudimentary which are the foundations for the later conduct, ha and speech. Patients are admitted to the Bicêtre a the age of two years and over.

In order to understand the methods of pedagogic tre

4. To teach the patient the use of language.
5. To arouse the intellect by inculcating ideas of length, weight, surface, solids, form, number.
6. Finally, to carry the education higher, by means of studies in natural history and all sorts of manual and industrial and moral training.

Naturally, some of these purposes are attained at the same time to a considerable degree by some one process employed in education. Thus, when a light bean-bag is thrown at the face of our patient, the attention and sensibility may be so feeble that it is not noticed at first. By frequent repetition attention is developed, sensibility becomes more acute, a reflex movement to ward off the missile is aroused, and gradually, by successive stages, the patient learns to catch the bag, to throw it back, and, finally, to go through a simple drill with it, accompanied by music. This single experiment then improves the attention and several of the senses, and aids in developing coördination and strength of the muscles.

Attention.—The degree of attention is, in the idiot, an indication of the degree of idiocy. To a certain extent the degree of attention noted is of value in prognosis; for, if the attention can not be aroused at all, no progress in education can be made. Thus the first step in our process of education must be the employment of methods of exciting attention. The most useful are such as appeal to cutaneous sensibility, to the eye, and to the ear. But even if these are in abeyance, the other senses afford useful avenues of approach to the nervous centers. Pricking, tickling, light blows, hot and cold articles, etc., may be used to attract attention through the skin. Colored balls, brilliant pieces of cloth, a ray of light in a dark room, the magic lantern, or a spectrum—such things may be variously and patiently experimented with to fix the attention of the eye. A loud call, a bell, music, a gong, or even a pistol shot sometimes, are devices for exciting the attention of the ear. Not infrequently months of patient experiment must be traversed before we are rewarded for our labors.

Education of the Sense of Touch.—The methods in vogue for developing the sense of touch generally aid at the same time the coördination of muscular movements; hence in actual practice the education of the hand and touch and also of the eye proceed more or less simultaneously.

The idea of temperature is developed by plunging the hand into cold, tepid, or warm water, or by the application of bottles containing water at different temperatures.

The sense of smoothness or roughness of surface is inculcated by passing the finger-tips over a board, one-half of which is covered with velvet, the other half roughened like a grater. Pieces of stuff of varying degrees of roughness or smoothness are also made use of. The softness and hardness of objects are taught by the handling of different objects, such as hard balls or cushions.

The child is taught to button by means of two bands of cloth, one with large buttons and the other with large button-holes; to lace up a shoe, by means of a shoe with eyelets a centimeter in diameter, and alternately hemmed with red and blue leather; to tie knots, with the aid of a pad upon which are spread strings of divers colors.

Stringing beads and buttons, sticking pins into a pincushion covered with dotted stuff, and the use of the size-board and form-board are useful means of developing tactile sense, educating the eye, and bringing out some of the faculty of calculation.

The Education of the Eye.—After the physician has remedied any existing visual defects, it becomes the duty of the instructor to interest the restless and inattentive eye. As already mentioned, the attention is aroused by glittering and striking objects, and, once the gaze is captured, the latent sense may be drawn out by many devices familiar to the kindergartner and teacher. Particolored balls, variegated shapes and colors of blocks, spheres, squares, cubes, illuminated pictures, gaudy stuffs, the spectrum, the kaleidoscope—all of these play a rôle in the education of the vision of the defective pupil. The matching of ribbons, wools, or cards, and the discrimination of forms of blocks, are methods of aiding the higher development of the visual sense. The size- and form-boards already alluded to, and the use of graduated rods to be placed by the pupil in step-like rows, are excellent adjuncts. Later on come into play various games,—dominoes, ball, croquet, marbles, bean-bag, hoops, tennis, skipping, battledore and shuttlecock, quoits, golf, and the like,—in the employment of all of which not only is the vision stimulated and improved, but there is a gain in manual dexterity, and an associated development of some of the psychic functions. The teacher acquires a special tact in leading the pupil to concentrate his mind upon what is being done, and in making use of the instinct of imitation, so that the child endeavors to do as the other pupils are doing or to follow the movements of the instructor.

Education of the Sense of Hearing.—After the physician has made sure that defective hearing is due rather to want of attention than to any of the many causes of deafness, the teacher experiments upon the sense with sounds of various kinds—gongs, bells, speech, instrumental music, and songs—and by some one of these means the ear will at last be reached and kept open until it becomes an avenue for impressions from the environment to travel to the brain for registration and the rousing of new cerebral activities. This organ in the defective is often especially alive to the influences of melody and harmony, to songs and jingles and rhymes. Music is an efficient aid in the various drills and games made use of later on in the child's mental development.

Education of the Taste and Smell.—While these senses have not the importance of the three just described, it is still useful to stimulate and develop them as far as possible. The child can be taught to discriminate between the simple taste sensations—salt, sweet, bitter, and sour—by means of solutions of salt, sugar, quinin, and citric acid, and between odors that are noisome and odors that are pleasant by means of tinctures of asafetida, cloves, and musk, and divers perfumes. Later, he learns to distinguish flavors, and to associate what is good and useful with pleasant, and what is hurtful with noxious tastes and smells.

Teaching to Walk.—A course of light massage of the lower extremities, together with exercise of the joints in flexion and extension, is undertaken for the purpose of developing suppleness and strength and improving the nutrition. The child is then regularly placed in a swing constructed for the purpose, with a vertical board in front in such a position as to receive the advancing feet of the child as it moves to and fro. The impact of the feet upon the board, with the backward swing caused thereby, in the course of time gives the child a sort of pleasure, and awakes in it a sense of the dependence of its movement upon the varying pressure and impact of its feet. It is not long before the child is enabled to use its legs with considerable ease and skill in the exercise. Having attained this stage, the child is now frequently held upright on its feet and then placed between the parallel bars sustained by its arms, in which position it is induced to make efforts at walking, at first for a few minutes, but with gradual increase of the time of stay each day. Then the pupil becomes quickly ready for a wheel-chair, which is merely a modification of the principle of the parallel bars, the supports being on wheels, so that as the child walks it moves the apparatus about with it. Later on it is taught to mount and descend a stair by means of a short, stationary step-ladder. After this the gait is rapidly improved by a variety of exercises, drills, simple dances, and the like.

Education of the Hands.—Even though the motions of the hands be incoördinate and without force, though the infant may be unable to do anything for itself, even to grasp an object or to oppose the thumb to the fingers, there are many methods of overcoming such defects and developing the normal power and usefulness of the hands. Among these is the employment of the parallel swinging-ladders and rings. At first the child's hands are applied to the rounds and held there by the teacher during the execution of such movements as standing, sitting down, raising the arms high above the head, and bending forward and backward, swinging to and fro, and so on.

As the pupil makes progress, the drill is carried on with great regularity and precision, accompanied by spoken commands and often with music. In this way not only are the muscles strengthened and coördinated and the use of the hands and feet perfected, but a familiarity with certain words and ideas and their association is created.

The use of blocks in building up various structures, with the subsequent pleasure of tumbling them down again, is as useful to these defectives as to normal infants.

Finger-exercises with the peg-board, or by means of picture-perforating, as practised in the kindergarten, may come into play for the development of the finger movements of the hand. Some of the apparatus employed in educating the sense of touch are equally valuable for training the accurate movements of the hands.

Teaching Habits of Cleanliness in Person and Dress.—Idiots of every degree are slovenly, awkward, negligent, unless taught and supervised, and the lower grades are incompetent to use spoon, knife, or fork, unable to care for themselves in any way, and continually drooling, sucking their fingers, holding the mouth open, and wetting and soiling themselves. It is of paramount importance, then, in their education to make every effort to overcome these deficiencies.

Such children as are unable or just learning to walk are placed by day in especially constructed chairs, and by night in especially prepared beds, for purposes of cleanliness, and must be watched and raised at certain hours by the nurses. It is surprising how many will, by assiduous attention, soon learn to give some signal to the care-takers of their needs, and in the end acquire control over themselves in this regard. They learn to expect the regular bath, and those who progress further become systematic in ablutions, cleansing the teeth, and all the little matters pertaining to the toilet. At the table they are taught first the use of the spoon, then of the fork, and lastly of the knife. They learn to dress themselves and to make themselves neat and tidy, and ultimately to brush and arrange their clothing, blacken their shoes, make their beds, etc. All of this instruction requires time and the utmost perseverance and patience on the part of the attendants. By it we also train the hands, the senses, and the intellect.

To close the mouth and prevent drooling, faradic electrization of the orbicularis oris is employed, and the insertion of a flat piece of wood or a stick of licorice-root in the mouth is useful. The teeth need careful looking over by a dentist from time to time, and daily cleansing. Sucking of the fingers and biting of the nails can be overcome by application of aloes and other bitter or disagreeable substances.

The Teaching of Language.—In idiots we must begin our inculcation of the uses of language according to the laws of its evolution in the normal child, first, however, correcting such defects in the ear, mouth, or vocal apparatus as are amenable to medical or surgical treatment. A child first develops its auditory word-center and then the motor speech-center. These two centers, with an association tract, are the primitive basis of language in the child. Often, in defective children, a course of gymnastic exercises of the lips, tongue, and jaw will be a necessary adjunct to the instruction, and in cases of deafness the lip-imitation method of education will require to be used.

In developing the motor speech-center the child begins by repeating the simplest linguals and labials, such as "dadda," "tatta," "mamma," "papa," and "babba," and these first consonants should be employed in the construction of the new words to be learned.

Music is an excellent auxiliary in teaching the articulation and use of words, and Shuttleworth recommends Elliott's "National Nursery Rhymes," set to pleasing melodies, as particularly adapted for the purpose. The interest of the pupil is often best secured and sustained by the employment of objective illustrations. The naming of subjects of pictures, of persons and things about the room, of parts of the body, and the imitation of cries of animals, are means of arousing interest.

After developing the word-hearing and the motor speech centers, the visual and writing centers will require education, and the methods in vogue are analogous to those of the kindergarten. Bourneville recommends the use, first, of black letters twelve centimeters high; then an alphabet with the consonants in black and the vowels in red, the letters six centimeters in height; then letters of ordinary size; and, finally, the repetition, in chorus, of letters and words placed before a class. This collective exercise, in which imitation plays a great part, contributes markedly to the development of speech. Figures are employed in much the same manner, and counting is learned from some of the various apparatus already described, as well as from simpler and more interesting devices, such as the use of the fingers, shells, marbles, buttons, beads, and the abacus. The nursery game of keeping shop is especially useful for developing the ideas of number, weights, and values.

Writing and drawing are taught by means of sand-boxes, blackboard exercises, and, finally, drawing-books. The knowledge of form is best inculcated by modeling in clay, and by reproductions in clay or wood of surface drawings.

From these primary lessons it is but a step to

Manual and Industrial Training.—When the pupil has reached a certain stage of mental development, every effort is made to further the training to such an extent as to subserve the demands of health and utility. Methods of manual and industrial education are best furthered in institutions in which every variety of occupation commensurate with the individual needs and tastes of the pupils can be satisfactorily carried out. In most existing institutions it is true that the ideal system of care and development of defectives has not yet been attained, but the tendencies of the present time are in the right direction. The institutions of the future for all classes of dependents, for idiots, for the insane, and for the inmates of prisons and reformatories, will doubtless be modeled on the colony plan. They will be village settlements or communities wherein the chief industries will be such as relate to the housing, clothing, feeding, etc., of their inhabitants, thus bringing into existence all of the occupations which tend to utility and economical administration. The scheme is well exemplified and successfully demonstrated by the evolution of the Craig Colony for Epileptics at Sonyea, N. Y. Were I called upon to draw up an outline of a plan for a colony for idiots, it would be somewhat as follows:

1. In the first place, there should be an abundance of land, at least an acre for each inhabitant. The site should be selected with due regard to fertility of soil; for agriculture, stock-raising, and gardening should afford employment for the majority of the pupils.

2. Convenience of access to managers and patients and their friends is a desideratum.

3. In the construction and arrangement of buildings the country-village idea should never be lost sight of, and the farmstead group—the cottages, villas, schools, shops, and so on—should be simple, independent, homelike, and surrounded by their own little gardens, hedges, etc., in conformity with such design.

4. So far as possible, each house should constitute a home circle, the number of members being limited to ten or fifteen.

5. An administration building, a small hospital for the sick, special villas for the infirm, bed-ridden, ineducable, and disturbed classes, a gymnasium, a library, a museum, and swimming- and rain-bath, are among the separate structures required in addition to those already mentioned.

6. The educational features of the colony will be carried on in ordinary schools, Sloyd schools, trade schools, and so on, and everything that may contribute to the furtherance of mental development should be encouraged. Thus the field study of natural history is one of the most satisfactory means of arousing the intelligence, interest, and activity of the pupils. Trees, garden produce, and flowers should be labeled with their names, botanical and zoölogical gardens should be established, and the collection of rocks, leaves, plants, insects, birds, etc., made a part of the system.

7. In developing the industries of the colony, such should first be instituted as will serve economical purposes. The aim should be to produce most of the foodstuffs required, to carry on domestic work, to make and mend the wearing apparel, to accomplish ordinary repairs, to construct new buildings, and to fashion the furniture.

8. The whole scheme requires to be under medical supervision, and the scientific aspects of the community thus created should be kept continually in mind. This necessitates the establishment of psychological and pathological laboratories after the most approved style.

As an instance of what species of work may be done by defectives in institutions, Bourneville's statistics of occupations at Bicêtre for 1897 show that there were 187 children employed in the various shops and workrooms, among them being: 10 brushmakers, 24 carpenters, 9 printers, 14 locksmiths, 51 tailors, 28 shoemakers, and 14 straw- and cane-workers. The hemiplegics work exclusively at sewing, and the blind with straw and cane. The colony plan, however, would insure a greater amount of healthier work out of doors than is possible at such a place as Bicêtre, and would be more remunerative to the administration.

Moral Training and Discipline.—Much as the inculcation of moral ideas is needed for normal children, defectives require even more attention in this respect; for in them the abrogation of higher intelligence is associated naturally with feeble inhibitive power. Thus they easily give way to the lower instincts, and are prone to acquire vicious habits of conduct and speech. In some cases the moral obliquity is so great that it constitutes the so-called moral imbecility, and little can be accomplished for their improvement. But the majority of defectives are susceptible to the influences of a good environment and moral discipline. Imitation of the teacher and of playmates and schoolmates counts for much with them. The judicious instructor and care-taker can, by firm and kindly guidance, accomplish great good in this respect, and it should always be kindly guidance, never coercion. There is, however, merit in the employment of a system of rewards and punishments adapted to the idiosyncrasies of the different pupils. A few words of encouragement or praise, or trifling compensations in the way of extra allowances of food, delicacies, recreations, or small wages, appeal distinctly to some; while words of disapproval, the curtailing of things pleasant to the palate, the deprivation of some anticipated pleasure, and so on, have especial influence with others. It is a good plan to distinguish the pupils for meritorious conduct and industrial accomplishments by distinctive dress, thus appealing to their ambitions. It is well to establish three or four grades to be thus distinctively recognized, for nothing is more human than the instinct to appear well to others, to be among the best-dressed. The instinctive desire of the savage for ornament is no stronger than that of the most civilized being for good clothes. The mentally feeble are no strangers to this feeling, and their good conduct can be enhanced and maintained by promotion to a better clothed division, and their shortcomings well punished by reduction to a lower rank. Corporal punishment is both necessary and useful in extreme cases with vicious tendencies, but should be a last resort even here.

By the means just described, and by other devices that will suggest themselves to the wise and tactful person whom we suppose to be intrusted with their care, these unfortunates may be taught obedience, perseverance, responsibility, and regard for the rights of others, and be imbued with some knowledge of the great laws of justice, beauty, goodness, and religion which rule the ideal world of humankind.

Physical Culture.—The tendency to incorrectness of gesture and bearing, the great lack of strength and grace, among idiots, must be overcome by systematic education of the muscles. There should be courses of gymnastic exercises and drills, with song and instrumental accompaniments. The drills may be made with wands, light dumbbells, etc. Military drill is excellent for both girls and boys. Dancing is beneficial to both mind and body. Bourneville has introduced fencing at Bicêtre, but does not speak of it with enthusiasm.

The Medical Treatment of Idiots.—The medical and surgical treatment of the different forms of idiocy is thoroughly discussed under their several chapters in this book, and it is intended here to refer only to the treatment of certain general conditions met with in all classes and grades of idiocy. Among such conditions are some that relate to hygiene, and others that pertain to bad habits, general diseases, and the like.

Hydrotherapy.—The rain-bath is nowadays considered a necessary adjunct to all public institutions, because of expedition in its use and perfect cleanliness. Such baths should be the daily morning rule of defectives. The skin is kept in a hygienic state, the circulation is stimulated, and general nutrition is improved by the morning bath. In lethargic or apathetic states the cold spinal douche is beneficial, while in very restless patients the prolonged warm bath and wet-packs at night often materially aid in overcoming the condition.

Clothing.—One of the noteworthy stigmata of degeneration common to all classes of idiocy is a diminished resistance to external influences and diseases. They catch cold easily. Tuberculosis and other lung disorders account for nearly seventy-five per cent. of the mortality among them. Diarrheas are common. Hence it is important that, among other things, considerable attention should be given to clothing. Woolen undergarments of warm and light texture should be the rule. The outer clothing should be light, durable, neat, of prevailing cuts and styles, and none of the clothing should in any way impede or restrict the free motions of the limbs and trunk.

Food.—The dietary for this class of defectives should, in my opinion, closely approximate that of epileptics—*i. e.*, it should be chiefly vegetable, with the free use of milk and eggs, and meat but once daily. Simplicity of food and simple cooking are essential. The dietary need not be so elaborate as, for instance, in hospitals or asylums, where acute disorders are commonly treated, and where the percentage of cure is expected to be large. Idiots are apt to overeat, and hence the chief requisite is to regulate the *per capita* allowance to just the amount necessary to maintain a robust state of physical health. Overeating is probably responsible for much of the diarrhea commonly observed among these cases.

General Bodily Health.—Very common is a condition of general debility, which must be met by appropriate tonics, nutritive foods, special baths, massage, and regular exercise. The great mortality from tuberculosis should lead the physician to a regular examination of the viscera for symptoms of that disorder. When discovered, the usual precautions should be taken to isolate the patient and to build up the constitution in every way. Parasitic and nervous skin diseases will often need attention. The prevailing mucous diarrheas are treated by the usual remedies and by careful regulation of the kind and amount of food. Owing to feebleness of constitution and diminished resistance to diseases, especial danger attaches to acute infectious fevers in idiots.

Masturbation.—The prevalence of this pernicious habit among all classes of idiots is only too pronounced. In the lowest grades it is uncommon, but among the imbeciles and feeble-minded it is one of the most intractable of conditions. There are few agents and devices which have not been tried, and usually vainly, to prevent the practice. It is only rarely that vesication of the genitals, punishment, mechanical restraint of the limbs, and sedative drugs have any effect in the treatment of defectives. Indeed, they might usually as well be left untried. There have been very few experiments of the method of cure by castration, for, naturally, professional opinion is too conservative to undertake, without long and careful deliberation, so radical a remedy. I know of but one institution where castration has been apparently adopted as a part of the regular system of care and treatment. The superintendent of the Winfield, Kansas, Asylum for Idiots has had between twenty and thirty boys who were inveterate masturbators subjected to castration, with excellent results. Not only were their vicious habits put an end to, but there was marked physical improvement in all, and great mental improvement in most, of them. There would seem to be no reasonable objection to operative procedure in such cases, though, perhaps, it is hardly necessary to go so far as castration. Ligature of the vas deferens, or possibly section of some branch of the pudic nerve, might serve as well. At any rate, some method of this kind is well worthy of consideration, though the ultimate decision of the profession as to its utility and propriety has yet to be learned.

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